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THE CARDINAL MANIFESTATIONS OF PAROXYSMAL TACHYCARDIA*

I. Anginal Pain

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THE symptoms accompanying paroxysmal tachycardia are numerous and varied. Most of them are not caused directly by the tachycardia, nor are they directly or specifically related to the underlying heart condition. Inquiry into the circumstances under which they arise throws no light on their meaning or on the underlying cardiovascular status. A few of the symptoms, however, arise under such constant conditions that they create a uniform clinical pattern. Because these symptoms — palpitation, pulmonary congestion, anginal pain, congestive failure, vascular collapse, central-nervous-system manifestations and embolism — may be correlated with significant variables, such as the type of patient, the heart lesion, the functional status of the heart and circulation, the ventricular rate and the presence or absence of embolism, it is proper to designate them as the cardinal manifestations of paroxysmal tachycardia. A detailed clinical analysis of 125 cases served as the basis for these and other conclusions, which were reported in a previous publication.[1] The present series of papers is devoted to a consideration, in greater detail, of some of the cardinal manifestations previously discussed. To the first group of 125 cases have been added 128 unselected new ones, making a total of 253. All cases had an electrocardiogram, confirming the presence of an abnormal rhythm and providing the ventricular rate during the paroxysm.

NONANGINAL CHEST PAIN

Chest pain is usually mentioned as one of the symptoms associated with paroxysmal tachycardia, but its incidence, character and significance are variously estimated or entirely disregarded by different authors. In 1888, in a paper containing the first clear description of paroxysmal tachycardia, Bristowe[2] stated that 1 of his 9 patients had angina-like attacks during the paroxysms of rapid heart action; similar observations have been reported by other authors[3–6] in single case reports. Barnes and Willius[7] noted the occurrence of anginal pain in 19 of 380 cases of paroxysmal tachycardia; the pain was usually described as a precordial ache; in no case did angina pectoris occur between the paroxysms. In 4 cases reported by White and Camp[8] and in some of those previously reported by me[1, 9] there was angina pectoris on effort; the former authors considered the induction of anginal pain by paroxysmal tachycardia a rare disorder. Campbell and Elliott[10] stated that tightness in the chest was present in most attacks of paroxysmal tachycardia and was of no special significance; in 12 of their 100 cases there was anginal pain, which was considered benign because induced by paroxysmal tachycardia. Williams and Ellis[11] recorded the occurrence of severe substernal pain in 2 patients during paroxysmal ventricular tachycardia; both patients had large hearts but were free of angina pectoris between paroxysms. The first study in which the induction of anginal pain by paroxysmal tachycardia was correlated with specific cardiovascular conditions was reported from this clinic in the paper referred to above.[1]

Analysis of the present larger series of cases shows that anginal pain was present during paroxysms of tachycardia in 40 cases, or 16 per cent of the series; these will be discussed in detail below. An almost equal number of patients had pain that was not anginal in character. In view of the significance of anginal pain in paroxysmal tachycardia, it is necessary to make a clear distinction between it and other types of pain. Nonanginal pain is frequently described as a precordial ache or distress, during or following the cessation of paroxysms of tachycardia. This is particularly frequent in patients with mitral stenosis, but is not limited to this group. Other patients are conscious of a tired or weak feeling in the region of the heart. Occasionally the cause of pain is the palpitation itself. This painful palpitation is usually seen in sensitive persons; consciousness of the heart action is interpreted by them as pain and is extremely disturbing; as the

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paroxysm ends there may be a severe, sharp, momentary exacerbation of the pain. Another type of nonanginal pain associated with tachycardia is sharp, pleuritic-like pain, often of great severity, over the precordium and left shoulder, occurring with inspiration. Upper abdominal pain due to acute congestion of the liver from an exacerbation of congestive failure secondary to paroxysmal rapid heart action may suggest anginal pain but should be differentiated without difficulty. Some patients complain of momentary twinges of precordial pain during paroxysms of tachycardia, and as a rule, similar pains are experienced between paroxysms; in other patients, vague, unexplained chest pain is noted during tachycardia. There is little to suggest anginal pain in these cases. In patients with neurocirculatory asthenia, pain is frequent with paroxysms of tachycardia, as it is during the normal cardiac mechanism. Another type of pain is so-called "imitation angina," occurring in those familiar with the syndrome of angina pectoris, such as physicians or nurses.

The remaining examples of nonanginal pain associated with paroxysmal tachycardia have no relation to heart disease or are not at all related to the tachycardia. The most important of these are pulmonary embolism and pericarditis.[12] In this series other causes of pain simulating angina pectoris and associated with paroxysmal tachycardia were peptic ulcer, gall-bladder disease, chest trauma or fractured ribs, neoplastic disease in the chest, postoperative pain in the upper abdomen or lower chest, splenic infarct and spontaneous pneumothorax. Pain, at times simulating angina pectoris, may occur in all these conditions, which may be complicated by paroxysmal tachycardia; the pain is due to the underlying condition and not to the tachycardia.

CASE 1. S. S., a 25-year-old man, had painless paroxysmal auricular fibrillation, but careful study revealed no evidence of organic heart disease. After exercising in a gymnasium, he was seized with severe, constricting precordial pain. An electrocardiogram showed auricular fibrillation with a ventricular rate of 120. Investigation revealed that the pain was due to spontaneous pneumothorax.

ANGINAL PAIN

Pain with the features characteristic of angina pectoris was classified as anginal pain regardless of duration. Since the pain in these cases was induced by tachycardia, the ordinary inciting causes of effort and emotion were absent. Most of these patients also had angina pectoris with effort or emotion; the similarity of the pain induced by paroxysmal tachycardia to that occurring at other times facilitated its interpretation. The duration of the pain depended on the duration of the tachycardia and the coincidental occurrence of acute myocardial infarction. With very short paroxysms the pain was identical with an ordinary attack of angina pectoris, but with prolonged tachycardia the pain was intermittent (status anginosus) or continuous, simulating acute myocardial infarction. The latter term is used in its widely accepted clinical sense, but includes major coronary attacks with evidence of heart-tissue necrosis. A diagnosis of acute myocardial infarction was made in 10 of the 40 cases having anginal pain.

Pain in the Absence of Acute Myocardial Infarction

Anginal pain in the absence of myocardial infarction was induced by paroxysmal tachycardia in 30 cases. The ages of the patients in this group ranged from twenty-eight to eighty-five years; there were 5 cases in the fifth decade, 9 each in the sixth and seventh, 5 in the eighth and 1 in the ninth. The single 28-year-old patient had rheumatic heart disease and free aortic insufficiency. There were 15 cases of paroxysmal auricular fibrillation (50 per cent), 9 of paroxysmal auricular tachycardia (30 per cent), 4 of paroxysmal auricular flutter (13 per cent) and 2 of paroxysmal ventricular tachycardia (7 per cent). The ventricular rate was 150 per minute or more in 26 cases, 140 in 3 and 120 in 1. Twenty-two patients gave a clear history of angina pectoris on effort, but no such history could be elicited in the remaining 8 cases. Of the latter, 3 developed acute myocardial infarction one to seven days after paroxysmal tachycardia had induced anginal pain. One patient had free aortic insufficiency; another had had an acute myocardial infarct six years previously, but no angina pectoris for a year preceding the induction of anginal pain by paroxysmal ventricular tachycardia. One patient in this group was a seventy-five-year-old woman with hypertensive heart disease and crippling arthritis. Only 2 patients had apparently normal hearts. Thus, of the 30 patients in whom paroxysmal tachycardia induced anginal pain, 73 per cent had exertional angina, 10 per cent did not have angina pectoris but developed acute myocardial infarction within a few days after paroxysmal tachycardia-induced anginal pain, 10 per cent had organic heart disease, and 7 per cent had normal hearts.

The following 4 cases are typical examples of the induction of anginal pain by paroxysmal tachycardia.

CASE 2. I. O., a 65-year-old man, had had angina pectoris on effort for 3 or 4 years, always relieved by nitroglycerin. While in the hospital he had repeated attacks at rest, always associated with paroxysmal auricular tachycardia with a ventricular rate of 170. The pain lasted continuously during the tachycardia and was not relieved by nitroglycerin, but ended at once when the abnormal rhythm was terminated.

CASE 3. S. W., a 58-year-old man, had a history of old myocardial infarction, paroxysmal dyspnea and angina pectoris on effort. During a routine examination he had repeated attacks of angina pectoris, weakness and faintness. It was noted that the episodes of pain coincided with extremely rapid heart action, and that when the heart rate suddenly slowed spontaneously or as the result of gentle stimulation of the right carotid sinus, the pain immediately disappeared. An electrocardiogram showed paroxysmal auricular tachycardia with an auricular rate of 214 and intermittent 2:1 auriculoventricular block, with a resulting ventricular rate of either 214 or 107.

Case 4. S. G., a 66-year-old woman, had had paroxysmal tachycardia for 50 years, associated with vascular collapse. Ever since the menopause 13 years previously, she had had hot flashes and the attacks of paroxysmal rapid heart action had increased in frequency. For several years effort provoked substernal, squeezing pain radiating into the back. Similar pain was induced during the same period by the paroxysms of tachycardia. When seen during an attack of pain the patient was in collapse, with a pale, moist skin, absent radial pulsations and a blood pressure of 76/70. The clinical picture was that of acute myocardial infarction. The heart rate, however, was very rapid, and an electrocardiogram showed paroxysmal auricular tachycardia with a ventricular rate of 200. Stimulation of the right carotid sinus interrupted the abnormal rhythm, with immediate cessation of the pain and return of a normal pulse and blood pressure. The patient was seen in several similar attacks, and at no time was evidence of myocardial infarction obtained. She died suddenly some months later.

Case 5. M. T., a 62-year-old male with a negative cardiovascular history, had a partial gastrectomy for a penetrating ulcer of the stomach. During convalescence he had a paroxysm of auricular flutter with a ventricular rate of 170; no symptoms were provoked, and he was entirely unaware of the paroxysm. Within 24 hours a second paroxysm with a ventricular rate of 166 occurred, but that time it induced anginal pain; when the paroxysm ended, the pain disappeared. On the following day, he developed severe, prolonged anginal pain, although the heart rhythm was normal. This was followed by elevation of the temperature and white-cell count and serial electrocardiographic changes indicating the presence of acute myocardial infarction.

Pain Associated with Acute Myocardial Infarction

The ages of the 10 patients in this group ranged from fifty-five to eighty-five; there were 2 cases in the sixth decade, 5 in the seventh, 2 in the eighth and 1 in the ninth. There were 4 cases of paroxysmal auricular fibrillation, 3 of paroxysmal auricular tachycardia, 2 of paroxysmal ventricular tachycardia and 1 of paroxysmal auricular flutter. The ventricular rate was 150 per minute in 5 cases and 154, 166, 170, 180 and 200, respectively, in the others. The onset of the arrhythmia and acute myocardial infarction appeared to be simultaneous in 4 cases; the tachycardia in 2 of these ended spontaneously after some hours, but the pain persisted, whereas in another the pain disappeared several hours after the attack began despite the continuance of paroxysmal auricular tachycardia for another twenty-four hours. Paroxysmal ventricular tachycardia with a ventricular rate of 170 occurred in 1 case on the third day of acute myocardial infarction, but did not appear to increase the pain, which was still present; two weeks later, after there had been no pain for over a week, paroxysmal ventricular tachycardia, with a ventricular rate of 170 to 180 recurred, but did not induce pain. Paroxysmal tachycardia in 3 cases occurred on the second to fourth days of acute infarction, while pain was still present but apparently without influencing it. On the sixth day after the onset of acute infarction in another case, intermittent anginal pain was still occurring, and was also induced by a paroxysm of auricular tachycardia with a ventricular rate of 150. The tenth patient continued to have spontaneous angina pectoris for four weeks following acute myocardial infarction, and paroxysmal ventricular tachycardia on the tenth and twenty-fifth days, respectively, induced anginal pain.

Case 6. J. H., a 70-year-old woman, had a history of diabetes, hypertensive heart disease and a previous cerebral accident. Her first attack of precordial oppression, accompanied by palpitation, a rapid heart rate and dyspnea, necessitated a hypodermic injection of morphine sulfate. On the following day the symptoms were still present and she was admitted to the hospital, where an electrocardiogram showed paroxysmal auricular tachycardia with a ventricular rate of 166. Three hours after the subcutaneous injection of morphine sulfate the pain disappeared, but the paroxysmal auricular tachycardia, without any change in rate, continued for another 24 hours. The data clearly indicated the presence of acute myocardial infarction.

Case 7. S. A., a 75-year-old woman with an apparently normal heart, had had for 5 years prolonged attacks of paroxysmal auricular tachycardia in which the only symptom was palpitation. She then had an attack of pressure in the chest, quickly went into shock and developed pulmonary edema. Examination showed paroxysmal auricular tachycardia with a ventricular rate of 200. The tachycardia stopped after 4 hours, but pain and shock continued for 4 to 5 days. The electrocardiographic and other evidence indicated the presence of acute myocardial infarction. Following recovery she had angina pectoris, both on effort and with paroxysmal auricular tachycardia.

Failure of Paroxysmal Tachycardia to Induce Anginal Pain in Coronary Heart Disease

Paroxysmal tachycardia failed to induce anginal pain in 48 cases of coronary heart disease. All the patients in this group had a past or present history of angina pectoris or myocardial infarction or postmortem evidence of significant coronary-artery disease. There was 1 patient in the fourth decade, three in the fifth, 12 in the sixth, 17 in the seventh, fourteen in the eighth and 1 in the ninth. Twenty-four (50 per cent) had paroxysmal auricular fibrillation, 12 (25 per cent) paroxysmal ventricular tachycardia, 9 (19 per cent) paroxysmal auricular flutter and 3 (6 per cent) paroxysmal auricular tachycardia. The ventricular rate was 150 or more in 32 cases and less than 150 in 16 cases.

Ventricular Rate Less Than 150

Of the 16 cases in this group, paroxysmal tachycardia complicated acute or recent myocardial infarction in 9. Paroxysmal auricular fibrillation with a ventricular rate of 120 per minute failed to induce anginal pain in a patient who was still having intermittent pain following a recent infarct. In 5 patients with a past history of angina pectoris on effort, paroxysmal tachycardia on the sixth, fourth, tenth, fourteenth and fifth days, respectively, following the onset of acute myocardial infarction failed to induce anginal pain, nor was pain occurring otherwise in these patients. The 3 remaining patients with acute infarction were asymptomatic (without pain) when paroxysmal tachycardia on the third, fourth and seventh days, respectively, failed to induce anginal pain.

Three patients with angina pectoris on effort had no pain during tachycardia with ventricular rates less than 150. The remaining 4 patients did not have exertional angina and pain was not induced by tachycardia; 2 had a previous history of myocardial infarction, and in the other 2 post-mortem examination disclosed the presence of significant coronary disease.

Ventricular Rate 150 or More

Thirty-two patients with coronary heart disease did not have anginal pain during paroxysmal tachycardia with a ventricular rate of 150 or more. Acute or recent myocardial infarction was present in 18 cases, in each one of which the tachycardia occurred when the illness had become painless. The tachycardia occurred on the following days after the onset of acute infarction: in 1 case on the third day, in 3 on the fifth, in 1 on the sixth, in 1 on the eighth, in 4 on the tenth, in 1 at the end of two weeks, in 2 at the end of three weeks and in the remaining 5 after five to eight weeks. Seven patients had had angina on effort for variable lengths of time preceding the onset of acute infarction.

There remain in this group 14 patients in whom paroxysmal tachycardia with ventricular rates of 150 or more did not induce anginal pain. Only 3 of these patients had a history of effort angina, which, however, had disappeared many months before the onset of paroxysmal tachycardia; post-mortem examination disclosed significant coronary disease in 2 of these. Five patients had a history of old myocardial infarction but not of angina pectoris, and autopsy confirmed the diagnosis in the 1 patient who died. In the remaining 6 cases, angina pectoris or myocardial infarction had never been observed during life, but at autopsy significant coronary disease was found in all of them.

Discussion

The present study of anginal pain in relation to paroxysmal rapid heart action has confirmed many of the conclusions stated in the previous report[1] and has revealed other facts of practical interest. The critical ventricular rate for the induction of anginal pain is about 150; pain is not provoked below this level. Paroxysmal tachycardia invariably induces anginal pain in patients with angina on effort, provided the ventricular rate is 150 or more; the ventricular rate, not the type of rhythm, is the significant factor. With few exceptions, patients in whom tachycardia induces anginal pain have organic heart disease, mostly coronary heart disease and angina pectoris and occasionally aortic valvular disease; such patients rarely have normal hearts. When anginal pain is induced by paroxysmal tachycardia in patients who do not have effort angina, it suggests acute myocardial infarction, or it may be a premonitory symptom of infarction. It may also indicate disease of an entirely different nature, such as pulmonary embolism or pericarditis, in which the tachycardia bears no relation to the pain but is merely a complication of the underlying disease, which itself is responsible for the pain. These and other possibilities must be considered particularly when pain is present during tachycardia with rates well under 150 or when pain is associated with tachycardia for the first time with a history of preceding painless paroxysms. When the ventricular rate is less than 150, pain is generally not due to the tachycardia.

Pain was present in only 10 of the 37 cases of acute or recent myocardial infarction in this series. Of greater significance than the ventricular rate in these cases is the time of occurrence of the tachycardia in relation to the onset of infarction, or, more precisely, the fact that pain of infarction or intermittent, spontaneous anginal pain is or is not still occurring when the tachycardia begins. When tachycardia occurs early in the course of infarction, while continuous pain is present, it may or may not enhance the pain, which may even disappear while the tachycardia continues; but if, following infarction, spontaneous intermittent pain is occurring, the tachycardia invariably induces anginal pain, provided the ventricular rate is 150 or more. Once the constant or intermittent pain associated with infarction has disappeared, paroxysmal tachycardia, regardless of the ventricular rate, does not induce anginal pain.

In cases of asymptomatic coronary heart disease, paroxysmal tachycardia does not induce anginal pain. This group comprises those who never had angina on effort, those with a past history of myocardial infarction or angina pectoris and those with acute and recent myocardial infarction in the painless stage. It is obvious that failure of paroxysmal tachycardia to induce anginal pain does not exclude the presence of coronary heart disease.

Summary

The clinical data of 253 cases of paroxysmal tachycardia are analyzed with reference to the occurrence of chest pain.

Chest pain, anginal and otherwise, is a frequent symptom in association with paroxysmal tachycardia. Anginal pain occurs under constant conditions and is therefore one of the cardinal manifestations of paroxysmal tachycardia. It occurred in 16 per cent of the cases in this series.

Anginal pain is invariably provoked by paroxysms of rapid heart action in patients with angina pectoris, provided the ventricular rate is 150 (the critical level) or more.

In the great majority of cases in which paroxysmal tachycardia induces anginal pain, coronary heart disease, usually with angina pectoris, is present.

The association of anginal pain and paroxysmal tachycardia, with ventricular rates under 150 or regardless of the rate, in patients without a history of

angina pectoris suggests various diagnostic possibilities, both cardiac and noncardiac.

Paroxysmal tachycardia does not always enhance the pain of acute myocardial infarction.

The failure of paroxysmal tachycardia to induce pain does not exclude the presence of coronary heart disease, acute, recent or old myocardial infarction or angina pectoris in the past.

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A MODIFICATION OF AN OLD AND SIMPLE METHOD OF TREATING RECTAL PROLAPSE*

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UNFAMILIAR at the time with the literature on rectal prolapse, yet with a distinct impression that the various treatments employed were not consistently satisfactory, I tried in October, 1939, a method that I humorously named the "garden-hose" technic. I had a feeling of originality that I soon learned was only partially justified. As will become apparent, this method may hardly be classified under the heading of the art of surgery, but the results thus far, in view of the simplicity of the procedure, have been fairly good.

CASE 1. M. C., a 71-year-old, married woman, had a 12.5-cm. procidentia of the rectum. A section of ordinary garden hose 15 cm. long, with an external diameter of approximately 2.5 cm. and an internal diameter of about 1.8 cm., was chosen because of its noncompressibility. In it a circular groove 1.5 cm. wide was cut deeply 4.0 cm. from the end to be inserted into the bowel. The patient was narcotized with morphine and scopolamine. The rectal sleeve was dragged out its full length and carefully palpated for the presence of small intestine in the herniated pouch of Douglas. With the aid of lubricating jelly, the hose was inserted so that its groove came just distal to the anal margin. Compression of the bowel against the groove was accomplished by ligatures of doubled No. 2 chromic catgut and strong, narrow rubber bands. As a further precaution against too early expulsion of the hose, the end of the prolapse was clamped to its outer end. The operation was performed in 13 minutes.

The postoperative course was reasonably comfortable, although the patient was alarmingly febrile. On the 13th day, the hose and slough came away. She was discharged on the 23rd day. There have been no rectal complaints, and repeated examinations have revealed no abnormalities during the 4 years and 11 months that have elapsed.

CASE 2. S. T., a 67-year-old, married woman, had a 5-cm. prolapse. In November, 1940, an operation similar to that described in Case 1 was done, with short anesthesia by ether. Convalescence was short and uneventful. The hose was removed on the 6th postoperative day and the patient was discharged on the 12th day. After 1 year there was a recurrence 4 cm. long, and in September, 1942, the same operation was performed, along with a small plastic for exertional urinary incontinence, but at that time constriction was made by twisting copper wire of 1.5-mm. diameter around the mass with electrician's pliers. Succinylsulfathiazole was given preoperatively and postoperatively, and ether was the anesthetic. Convalescence was uneventful. The hose was expelled on the 11th postoperative day and the patient was discharged on the 15th day. She remained cured for 1 year and 6 months. Symptoms recurred in March, 1944, and there is now a recurrence that starts 3 cm. above the anus and protrudes for 3 cm. on straining. Eighteen centimeters of bowel has already been removed, and 12 cm. more soon will be. Perhaps one of the newer procedures by the abdominal route would be more effective,[1-4] but in view of the patient's age the risk of another recurrence in 1 or 2 years is preferable.

CASE 3. A. M. P., a 64-year-old, unmarried woman, had a 6-cm. prolapse of the rectum. In May, 1941, an operation similar to that in Case 1 was performed, with anesthesia by ether. Convalescence was uneventful, and the patient was discharged on the 19th postoperative day. A stenosis formed that required repeated dilatation during the following 4 months and has for the last 3 years given occasional slight trouble. The patient is satisfied with the result, and there has been no recurrence of prolapse.

CASE 4. E. C., a 69-year-old, married woman, had an 8-cm. procidentia of the rectum. She received succinylsulfathiazole preoperatively and postoperatively. In September, 1942, under short anesthesia by Pentothal Sodium, the same technic as that described above was employed, compression being accomplished by wire and pliers. The postoperative course was smooth. On the 11th day most remaining slough was removed by cutting against the hose, which was then withdrawn. The patient was discharged on the 15th day. During the last 2 years she has not admitted a complaint, but she has had a stricture 4 cm. above the anus. This was twice dilated in the fall of 1943. It now has a lumen 1.7 cm. in diameter.

CASE 5. M. J., a 51-year-old, married woman, had a 9-cm. procidentia of the rectum, 7 cm. in diameter. Disturbed by the strictures in the previous 2 patients, even though they were of minor inconvenience, I reasoned that they had occurred because of too short a diameter of the hose at the groove of compression. Therefore, instead of cutting a groove, I built up a circular trough as follows. First, the external diameter of the hose was increased to 3 cm. by winding broad adhesive plaster around it. On each side 4 cm. from the end to be inserted into the rectum 2-cm. adhesive

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plaster was wound, leaving a trough 1.5 cm. wide and 0.5 cm. deep.

The patient received succinylsulfathiazole preoperatively and postoperatively. A plastic operation for a lacerated perineum and a large rectocele with enterocele was performed in December, 1942, ether being the anesthetic, before proceeding with the "garden-hose" maneuver. Constriction was made with copper wire, the ends of which were left long and hooked into the outer lumen of the hose. On the 6th postoperative day most of the slough was cut away. Fecal obstruction of the large intestine was easily cleared by enemas given through the hose, which was removed on the 8th day. Convalescence was excellent, and the patient was discharged on the 14th day. She has been well for 1 year and 9 months, and there has been no narrowing of the rectal lumen or recurrent prolapse.

Four of these patients were elderly and frail and had cardiovascular disease; the fifth was obese. Until two years ago, catharsis and enemas were given as part of the preoperative preparation and defecation was inhibited by tincture of opium for ten days postoperatively. Succinylsulfathiazole has made this regime unnecessary and has apparently been the chief factor in achieving practically afebrile convalescences. The daily use of soap and water, boric acid powder and dry dressings was adequate for the care of the local area. All the patients had stretched, relaxed anal sphincters before treatment, and in all cases good muscular tone returned afterward. If one is not sure that the herniated pouch of Douglas is empty, there is no stringent contraindication to incising the anterior side of the rectal procidentia well below the anal margin and exploring before proceeding with constriction. I have not yet considered it necessary to do this.

Discussion

Older methods of achieving constriction amputation involved ligatures or elastic bands, and rubber tubing. The only original features of the present method are the cutting of a circular groove in garden hose or building up a trough around it and drastic compression by twisting strong malleable copper wire around the protrusion. Emerson,[5] in 1938, stated, "Generally a piece of garden hose . . . is used." Yet he is the only author to be found who specifically mentions garden hose.

Removal of rectal prolapses by constriction has been done for years, but not often, since any one surgeon's experience with this entity is usually limited to less than 10 cases. References to the early literature on removal by constriction are covered in papers by Moschcowitz,[6] Reid,[7, 8] and Eliason and Erb.[9] Reid stimulated renewed interest in this type of procedure, although he limited it to cases with irreducible prolapse. The ones I have treated were all reducible, as were the ones reported in the more recent literature. According to Eliason and Erb, only 1 death from this type of operation had been recorded — in 1900. They treated 3 cases in this manner with good results at four to fifteen months. Wangensteen[10] reported 1 case satisfactorily managed in this way with a good outcome two months later. Reid's 2 patients were cured between three and four years afterward. Anderson and Borland[11] had 1 case treated by the Reid technic with a good immediate result. In 1938, Emerson[5] recorded 2 deaths following treatment by elastic ligature and severe morbidity in 5 others, with ensuing stenosis. Thus, from the literature one would conclude that amputation of rectal procidentias by constriction is not a good procedure, since in the last twenty years there have been 2 operative deaths among 14 reported cases. There has also been considerable morbidity, as well as rectal stenosis, and the period of observation has been for the most part too short to decide about the permanency of cure.

A fairly complete review of the literature of the last fourteen years leads one to the same conclusion concerning the various other methods of treatment, both abdominal and perineal. Either the morbidity or the mortality is similar, or there is a high rate of recurrence, or good results are based on insufficient length of observation. This conclusion applies in part to my own cases, which are expected to increase and which will be followed for significantly longer periods.

The largest series of cases with apparently the best results is that of Miles.[12, 13] He performed surgical amputation of the protruded bowel on 34 patients, with 1 operative death and 1 recurrence at five years. Lockhart-Mummery[14] wrote, "For very large prolapses the best operation is amputation." Yet in 1932, Rankin, Bargen and Buie[15] stated, "Procedures directed toward the cure of the prolapse by resection have little to recommend them."

The objective of all methods of treatment is the formation of scar tissue between the rectum and the surrounding structures. This seems to have been well accomplished by surgical amputation and logically should be achieved just as well by compression amputation. The minimal loss of blood and the short time required are in favor of the latter technic, and succinylsulfathiazole has removed its greatest danger — namely, sepsis. The mortality and morbidity with the constriction method appear to have come from technical errors. For example, the hose has been inserted too far above the point of constriction, thus allowing the accumulation of feces between it and the rectum. Peristalsis may have forced some of this material through a weakened area in the tissues. There should not be more than a few centimeters of hose above the point of compression. Furthermore, it is probable that in some cases strangulation has not been complete, thus permitting much greater septic and toxic absorption. A slight increase in the diameter of the hose might have reduced or eliminated an undesirable amount of stenosis. From my experiences with uterine procidentias and vaginal relaxations, I am convinced that some patients have tissues so constituted as to be incapable of permanent anatomic

reposition, and that just as 15 per cent of gynecologic patients have more or less recurrence, so will there continue to be an unavoidable percentage of recurrent rectal prolapse.

Summary

Some modifications of an old method of removing rectal prolapses by compression amputation have been presented. In 5 cases of reducible rectal prolapse in women the procedure has been safe, and the results in 4 cases have thus far been good for periods varying from one year and nine months to four years and eleven months. In the fifth case, prolapse has twice recurred after twelve and eighteen months, respectively, of temporary cure.

A brief evaluation of the literature is included, with evidence that surgical amputation has yielded the best results. It is suggested that compression amputation should be just as effective, and it has the advantage of being a much simpler procedure.

8 Cumberland Avenue

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Discussion

Dr. James B. Woodman, Franklin, N. H.: Some years ago on looking up the Whitehead operation, which was in vogue at that time, I found that there was no apparatus described to put in the rectum as a form around which to do this operation, so I secured a champagne cork and fitted a heavy loop of wire in one end for a handle, introduced it and tied on heavy braided silk,— thus doing away with the necessity of putting tape there, — with excellent results. I have used this method three or four times since then.

Dr. Allen G. Rice, Springfield, Mass.: I cannot lay claim to the invention of any ingenious gadget, but five years ago next December an engineer presented himself in the office and demonstrated with the greatest ease what his trouble was. With hardly any exertion he could extrude about 20 cm. of his rectum. He told me that he had previously had three operations, one from below and two from inside the abdomen, and that all had been failures. He wanted to know if there was anything I could do. I asked for a day or two to think it over, and when he came back I proposed the following procedure, which he accepted. I opened the abdomen and pulled up all the sigmoid until it was taut, and then did a Mikulicz type of colostomy. Later I cut the spur and closed the incision. The patient has been perfectly well ever since.

Dr. E. Parker Hayden, Boston: Reid several years ago described 3 cases in which he carried out this procedure, but Dr. George Smith is the only person with whom I have discussed it firsthand. I am going to ask him if he had examined the slough to see whether by any chance it included the uterus, but he mentioned in the latter part of his paper that one could palpate and explore the cul de sac, if one desired.

I have had 1 case in which the uterus was palpable in the anterior wall of the prolapse — the entire uterus. When I inspected the abdomen I was unable to see the uterus in the pelvis until I reached down with my hand and pulled it up from the depths of the cul-de-sac herniation.

I looked these cases up at the Massachusetts General Hospital several years ago and found that we had had about 1 case a year. In a period of twenty-one years there were 20 cases, operated on by many surgeons and by a variety of methods, consisting principally of the Moschcowitz obliteration of the cul de sac. The mucous membrane, in some cases, had to be excised afterward because it persisted in prolapsing, and in several cases a plastic was done on the atonic sphincter. For example, one patient had had a complete prolapse for twenty years, and one year after doing both a Moschcowitz and a Lockhart-Mummery procedure, because of relaxed sphincter, I trimmed the mucosa away and did a plastic on the sphincter. Compression ligature is a simple method, and I do not see why it is not a successful way of handling this condition.

Dr. Reginald H. Smithwick has done several Delorme operations with considerable success. The mucous membrane is excised in a cuff around the prolapse, after which sutures are taken in the submucosa vertically, thereby reducing the length of the prolapse until the mucous membrane edges come together; these edges are then sutured.

One can apparently treat this condition successfully by a number of different procedures, of which the method described by Dr. Smith is undoubtedly the simplest technically.

RADIOULNAR SYNOSTEOSIS OF TRAUMATIC ORIGIN*

Report of a Case

HAROLD G. LEE, M.D.†

BOSTON

AN EXTENSIVE review of the medical literature reveals that there are few recorded cases of synosteosis of the forearm bones, although this is always mentioned as a possible complication in discussions of radial and ulnar fractures. The purpose of this report is to present a case of radioulnar cross-union that occurred as a sequela to comminuted fractures of the forearm bones, which were accompanied by a great deal of damage to the soft tissues.

A B C D

FIGURE 1. *Roentgenograms of Forearm.*

A — *This film shows the original semioblique fractures of the radius and ulna at different levels*; B — *this film, taken four days after open reduction, indicates that the fragments were in good alignment*; C — *this film, taken approximately two months after reduction, shows evidence of cross-union between the radius and ulna*; *and* D — *this film, taken about four and a half months after reduction, shows definite synosteosis.*

CASE REPORT

A large, muscular 44-year-old man, while at work in a factory on January 7, 1941, caught his forearm in a moving belt, sustaining an extremely severe injury. The radius and ulna were fractured, and the distal fragments with the hand were carried beyond a right angle by the force of the belt. Only by exerting great force was the patient able to prevent the forearm's being drawn into the pulley. Roentgenograms taken at the time of the accident revealed comminuted fractures of the radius and ulna, semioblique in type and situated 12 cm. above the wrist (Fig. 1*A*). The fractures were at slightly different levels, and the fragments were completely displaced.

In view of the severity of the trauma, it was realized at the outset of treatment that the soft tissues had undoubtedly been extensively damaged; hence, the possibility of the development of synosteosis was considered. The danger of circulatory disturbance was also considered, because of the sclerosis of the vessels that was revealed in the roentgenograms. To permit observation and ensure circulatory stabilization, the forearm was maintained in suspension in a molded plaster cast for 4 days.

Reduction by a closed method was not considered feasible because of the obliquity of the fragments. On January 11, I carried out an operative reduction, taking great care to prevent further traumatization. Two incisions were used for exposure. First, a longitudinal incision was made over the site of the radial fracture. The fragments were easily placed in normal anatomic alignment and made secure by applying a three-screw plate of vitallium. The membrane and periosteum were found to be badly lacerated, and small chips of bone were displaced into the interosseous space. An attempt was

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made to remove all the bony fragments and pieces of periosteum that lay between the forearm bones and to suture the torn soft tissues. Complete repair, however, was impossible because of the severe trauma.

The second incision, small in size, was made over the site of the ulnar fracture. It was found that the ulnar fragments had fallen into position on the reduction of the radial fracture, and it was necessary only to secure them with chromic catgut, which was drawn through two drill holes.

An uneventful convalescence ensued. Roentgenograms taken on January 15 showed the fragments in good alignment (Fig. 1*B*). The vitallium plate was removed in April.

The first definite evidence of cross-union between the radius and ulna was noted in a roentgenogram taken on March 7 (Fig. 1*C*). In subsequent roentgenograms the bridging between the bones became increasingly dense (Fig. 1*D*). Clinically, rotation of the forearm was impossible. Operative excision of the synosteosis was advised.

On October 11, the surgical treatment was carried out as follows. Under general anesthesia, an incision was made along the inner side of the ulna in the line of the synosteosis. The muscles were split down to the interosseous space and, with a great deal of difficulty, were separated from the bony growth between the radius and ulna. It was impossible to separate the muscles without carrying away some of the periosteum. The bridge of bone between the radius and ulna was next removed, thereby restoring the rotary motion of the forearm. The muscles on each side of the interosseous space were sutured in such a manner as to cover the raw bony surfaces. The forearm was supinated as much as possible, and a compression dressing was applied.

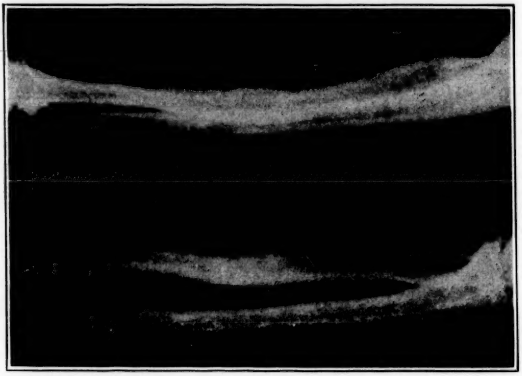

FIGURE 2. *Roentgenograms Taken Over Three Years after Resection of the Synosteosis and Showing No Evidence of Renewed Proliferation.*

The patient made a good recovery. Complete range of flexion and extension at the elbow was obtained early.

When the patient was examined on January 17, 1945, three years and three months after the removal of the bony bridge, he had recovered 75 per cent of the normal rotary motion at the elbow. There was slight limitation in the motion at the wrist, owing to the presence of arthritic changes. The hand had a complete grip. A roentgenogram taken on that date showed good integrity of the interosseous space (Fig. 2).

SUMMARY

A case of radioulnar synosteosis, with complete recovery following operative excision, is reported.

412 Beacon Street

MEDICAL PROGRESS

SCARLET FEVER*

CONRAD WESSELHOEFT, M.D.,† AND LOUIS WEINSTEIN, M.D.‡

BOSTON

WHEN Sydenham[1] coined the term "scarlet fever" in 1676, he remarked that this was a disease in name only. The truth of this statement becomes more and more apparent with the progress of knowledge of streptococcal infections. When the Dicks isolated their so-called "*Streptococcus scarlatinae*" and patented the toxin and antitoxin derived from it, the idea of a disease entity seemed to become more fixed. This patent has now expired. With the advance in the knowledge of streptococcal diseases many ideas regarding this eruptive fever have changed. Scarlet fever is not so much a disease entity as it is a manifestation in susceptible persons of a toxic substance elaborated by some strains of beta-hemolytic streptococci. Nor are the toxins elaborated from these types identical.[2] Not only can many different types of streptococcus produce a scarlet-fever toxin, but from the work of Aranow and Wood[3] it appears that certain strains of hemolytic *Staphylococcus aureus* can produce a typical scarlatiniform rash, and that they elaborate an erythrogenic toxin that is antigenically related to that produced by the streptococcus.

The sore throat of scarlet fever cannot be differentiated from any other streptococcal sore throat except that there is apt to be a bright-red enanthem on the soft palate. The eruption is much less likely to be on the face than is the case in measles and German measles. It is essentially an erythema due to engorgement of the superficial capillaries of the skin. Engorgement of the capillary tufts in the papillae gives rise to the punctate character of the rash. In a severe rash the capillaries tend to rupture, especially in the folds of the skin — the so-called "bleeding lines." The engorgement of the capillaries in the papillae of the tongue gives its strawberry or raspberry appearance. Except for the inflammation of the fauces, all these manifestations of scarlet fever are due to the action of the erythrogenic toxin on the superficial capillaries. This toxin is also responsible for the fever and the not infrequent nausea and vomiting and occasional diarrhea.

IMMUNITY

The Dick test is the measure of susceptibility to scarlet fever. It consists of an intradermal injection of 0.1 cc. of a standardized dilution of the toxin, with recording of the result twenty-four hours later. Those who are susceptible show a localized erythema and are recorded as Dick positive, whereas those who show no reaction are recorded as Dick negative. The Dick reaction is almost always negative at birth. Thus, an infant is born with immunity to scarlet fever but loses it rapidly as it approaches the second year of life.[4] From this age the percentage of Dick positives drops from nearly 80 per cent through the grammar-school period to somewhere between 40 and 20 per cent at the military age, according to the opportunity for exposure. This age curve of immunity as measured by the Dick test[4] is similar to the incidence curve of scarlet fever according to age.[5] Powers and Boisvert[6] have studied the factor of age in relation to the type of infection in streptococcosis. The incidence of other manifestations of streptococcal infections follows different patterns as regards age.

Generally speaking, the erythrogenic toxin of the many strains of streptococcus is antigenically much the same, and in the long run confers a lasting immunity. The immunologic response may be monovalent during convalescence and become polyvalent only well after recovery. In other words, the scarlet-fever patient sometimes reacts specifically to the type of organism with which he is infected. In this case, if he is exposed to another type during convalescence, he may have a relapse with a return of the sore throat, fever and rash.[7] On the other hand, given sufficient time, the immunity conferred by one attack usually affords protection against the toxin of a variety of strains — hence the relatively permanent immunity conferred by one attack of scarlet fever. Although the immunity conferred by a single attack of scarlet fever is primarily against the toxin, this does not of necessity include protection from subsequent bacterial invasion by the original type or any other type of streptococcus. Kuttner and Lenert[8] have shown that bacteriostatic properties develop in the blood of patients recovering from pharyngitis due to a single type of Group A streptococcus, and that these properties may persist for many months.

By injecting the erythrogenic toxin in repeated doses a relative immunity to scarlet fever can be achieved. This method of immunizing was begun in 1925. Unfortunately, the early dosage as printed on the preparations licensed through the Dick patent was too large and resulted in the very thing that the patent aimed to avoid, namely, bad effects from the toxin. Most pediatricians promptly discarded this procedure because it caused more illness

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than would have been expected from the usual scarlet-fever attack rate. By the time the directions on the package were modified the reputation of the Dick toxin in pediatric practice was bad, and its use has been continued only in the hands of a few practitioners.

The immunization of pupil nurses has been so satisfactory from the standpoint of economy that the procedure is carried out in most hospital training schools. Where this procedure has been carried out with the care and scrutiny due an erythrogenic toxin derived from streptococci, the discomfort has been at a minimum. With permission of the patent holders a formalized toxin was made by the Massachusetts Department of Public Health. This did away with the discomforts, but the immunity gained was not so good as that achieved with the straight toxin. Permission to continue this investigation in Minnesota was refused.[9,10] As far back as 1925, several hospitals were giving eight or ten doses instead of three or five, and thereby reducing to the minimum the incidence of sore arms and febrile reactions. A general hostility toward the patent prompted some institutions to make their own toxin for this purpose. Needless to say, their methods were not published, and an unknown amount of investigative work was stifled because of the difficulty of getting permission to perform it.

Toomey[11] has given an excellent review of scarlet-fever immunization. The attack rate of pupil nurses varied in different years in different hospitals from 8.6 to 17.7 per cent. Among the immunized nurses the attack rate in these hospitals was 0.6 per cent. Reactions were noted in 10.2 per cent. Pupil nurses are continuously and heavily exposed on the scarlet-fever wards. Prior to immunization it was usual to have several nurses constantly sick with scarlet fever throughout the winter months. It is now rare to have one nurse lost from duty from this cause. This economic gain to the hospitals for contagious diseases over the past twenty years has been enormous.

The question is raised whether this immunity is more apparent than real. In other words, it is argued that only an immunity against the toxin is achieved, and that these immunized nurses probably act as dangerous streptococcal carriers. On the other hand, it has been asserted that there is apparently somewhat less streptococcal disease among the immunized nurses as a result of this immunization. There is no substantial evidence to support either contention. In short, immunization against scarlet fever gives rise to a specific immunity against the toxin similar to that achieved by an attack of scarlet fever, probably no more and no less. It is distinctly worth while for pupil nurses whose training includes duty on scarlet-fever wards. Beyond this, there is room for debate owing to the low morbidity and low mortality of scarlet fever.

Recently scarlet-fever immunization has been instituted in the 25 per cent of men of the Royal Canadian Air Force[12] found to be Dick positive. The advantages and disadvantages of this procedure remain to be seen.

One disadvantage of immunization is that in those who harbor chronic streptococcal infections or who have had rheumatic infections in the past there is a sensitiveness to the hemolytic streptococcus toxin as manifested by polyarthritis, erythema nodosum and even heart disease.[13] Such persons, however, would show much the same symptoms if they came down with scarlet fever. Consequently, it is better for them not to be immunized and to avoid exposure to streptococcal diseases — if such a thing is possible. Therefore, in selecting those who are to be immunized, one should eliminate not only those who are Dick negative but those whose past histories offer contraindications.

Passive immunization has been used successfully in the last twenty-five years. Immediately after exposure a dose of pooled convalescent serum or Dick antitoxin is administered intramuscularly. The protection lasts only about fourteen days.

Forms of Scarlet Fever

It is necessary to keep in mind that the usual form of scarlet fever is the result of streptococcal infection of the upper respiratory tract. As such it is spread by direct and indirect contact, and it may also be air-borne. The dust in scarlet-fever wards is apt to be heavily laden with hemolytic streptococci, and the air is especially full of these bacteria after bedmaking and sweeping.[14] On the other hand, so-called "puerperal scarlet fever" results from a pelvic infection, and "surgical scarlet fever" from wound infection. Of course, the streptococci in these infections must be capable of producing erythrogenic toxin. "Burn scarlet fever" may come on at any time during the healing of an extensive burn when these hemolytic streptococci get onto the wound and elaborate their scarlet-fever-producing toxin. To these well-known forms of scarlet fever must now be added so-called "intestinal scarlet fever," which is due to the absorption of the toxin from the intestine in cases showing much gastrointestinal disturbance. The Dicks[15] showed that enteric-coated tablets of the toxin could give rise to vomiting and diarrhea, along with the rash. In this activity of the streptococcus in the gastrointestinal tract it may also give off an enterotoxin[16] that is quite apart from the toxin that produces the rash. The term "septic scarlet fever" is used when the pyogenic properties of the streptococcus are in evidence.

The Dicks[17] resurrected the term "*scarlatinae sine eruptione*" because in their inoculation experiments certain subjects became sick without the rash. The modern concept no longer grants any

such term. Without the rash there is no scarlet fever, even though the patient is infected with a streptococcus that is causing a rash in others. Furthermore, such a patient who does not have scarlet fever is capable of giving it to those who are susceptible. To include all infections of the toxin-producing streptococci as scarlet fever is a misuse of the term, since the resistance of the host is an equally important factor. Thus, as pointed out by Gordon,[18] scarlet fever is not just a problem of identifying a rash. From the clinical and epidemiologic standpoint, much depends on where the infection is situated, on the type of streptococcus and on the resistance of the host.

It is obvious that dissemination of these streptococci is greatest in those cases in which the infection is in the upper respiratory tract. In the puerperal form the infected discharge is fairly well confined by the pads over the vagina, especially when these are disposed of by the proper technic and bedpans are sterilized. The dressings of wounds and burns permit the least possible dissemination of infected material. Thus, with good modern surgical technic, a case of scarlet fever originating on a maternity or surgical ward rarely gives rise to a second case. Prompt isolation of the patient makes it unnecessary to quarantine the ward. On the other hand, when upper respiratory scarlet fever breaks out on a maternity or surgical ward, there is much more opportunity for the dissemination of the streptococci.

Treatment

Scarlet-fever antitoxin is the specific measure to combat the eruptive manifestation of this disease in that it neutralizes the erythrogenic toxin. Thus, the degree of toxicity manifested in the patient is the indication for this antitoxin, which may be administered intramuscularly or, in a severe case, intravenously, in the form of a polyvalent horse serum (Dick). In moderate cases, a patient under 50 pounds in weight should receive 8000 units, and in severe cases 16,000 units. In those over 50 pounds one should give 16,000 units in moderate cases and 32,000 units in severe ones. Pooled convalescent serum given intramuscularly or intravenously is also efficacious. Here a patient under 50 pounds gets 20 cc. in mild cases and 40 to 60 cc. in severe cases, whereas for those over 50 pounds the dose is 40 cc. in moderate cases and 60 to 100 cc. in severe ones. If given in the first forty-eight hours both these serums show a prompt and beneficial action. The temperature usually drops abruptly, the rash fades, and the sore throat subsides. Occasionally one sees no appreciable effect even with early treatment; in other cases dramatic response may be achieved as late as the fourth day. Serum therapy is unnecessary in mild cases, and is of no value in late septic complications.[19,20] The concentrated gamma globulin fraction from pooled normal plasma has been used in the eruptive phase, but judging from our observations the results in its present form are inconstant.

Sulfonamide therapy does not neutralize the toxin and therefore has no effect on the eruptive phase.[19–21] The temperature is not materially lowered, and the duration of the rash is not shortened. The sulfonamides do tend to control the further invasion of the streptococcus, and they definitely control the pyogenic activity of the streptococci if and when such activity is in evidence. In mild and moderate uncomplicated cases, when sulfonamides are given for the duration of the fever only, no appreciable benefit has been noted by most observers when adequate controls have been instituted.[22] In severe cases with evidence of deep invasion of the throat, serum therapy should be given along with sulfonamides.

If sulfonamide therapy is instituted at the start in adequate dosage to give a level of approximately 6 mg. for the first four days, and a maintenance dose of half that daily dose is continued for another four days and halved again for the next eleven days, the pyogenic complications are reduced. Nevertheless, one must not be surprised if suppurative otitis media and suppurative cervical adenitis develop in spite of this treatment.* Furthermore, drug rashes and drug fevers occur in certain patients to baffle the clinician, and in rare cases severe anemia develops rather suddenly and requires blood transfusions. In any event, the nonpyogenic complications are not avoided.

Complications

The complications of scarlet fever constitute the manifold expressions of streptococcosis. From the upper respiratory tract may develop sinusitis, especially ethmoiditis, which may be particularly prominent in certain epidemics. This, like suppurative otitis media, represents bacterial invasion with pus formation. The accepted therapy of all pyogenic manifestations is full dosage of sulfadiazine or sulfamerazine. If, after withdrawal of the drug, a discharge from the ear returns, it is well to consider the possibility of a mastoiditis. Here one must keep in mind that sulfonamide therapy may hold back a leukocytosis and mask the x-ray findings.[23] The postponement of surgery may delay recovery. Surgical mastoiditis has been greatly simplified by the local use of sulfonamide drugs, and more recently by the use of penicillin.[24] Severe tonsillitis, pharyngitis, laryngitis, pneumonia, empyema, meningitis and bacteremia call for intensive sulfonamide or penicillin therapy. In the presence of rheumatic fever, penicillin is to be preferred for the treatment of the suppurative complications.[25,26]

A generalized lymphadenitis is a frequent accompaniment of scarlet fever. This occurs early in the disease as a result of the circulating toxin. Later on, between the tenth and the twenty-first day, there may be a recrudescence of lymphadenopathy,

*The widespread use of sulfonamide therapy and prophylaxis may be responsible for the development of sulfonamide-resistant strains of streptococci during an epidemic of scarlet fever.

especially in the cervical region. The lymph nodes in the neck become swollen and tender, but if they are soft and not particularly painful no chemotherapy is indicated even though fever is present; nor are any local applications necessary. This situation represents a late reaction to the streptococcal infection, is manifested also in the axillas and groins, and subsides of itself. A hard, firm, extremely tender cervical node is quite another matter and may represent a pyogenic invasion. Here the question of chemotherapy is debatable. Chemotherapy undoubtedly controls the pus formation, but the induration remains for a long time. Sometimes it remains longer than if the sulfonamides had not been used and the node had been allowed to suppurate and point to the surface and had then been incised. The application of heat or cold has no apparent influence on the course of such suppuration, since pointing takes place under an ice bag as it does under a hot poultice. Either heat or cold may afford a definite degree of comfort.

In addition to the suppurative complications following scarlet fever, there are several others that are the result of some altered response to the causative streptococci or their products, namely, acute diffuse glomerulonephritis, erythema multiforme and rheumatic fever, the tissue changes of which are discussed by Mallory and Keefer.[27]

Nephritis in scarlet fever is of varying degrees of seriousness. During and following the eruptive phase an increased Addis count[28] is found even in the mildest cases. A mild transient albuminuria may also take place. These are both due to the irritant action of the toxin. When red cells or casts begin to show in an ordinary centrifuged specimen of urine, one must be on guard. The explosive nature of acute hemorrhagic glomerulonephritis suggests a violent antigen-antibody reaction in the glomeruli. This is apt to occur some time between the ninth and twenty-first days of convalescence. The urine on the previous day may have been quite normal. The situation is the same as that in glomerulonephritis in diseases other than scarlet fever and calls for the same treatment.[29,30] Glomerulonephritis cannot be prevented by dietary regulations.

Erythema nodosum, erythema marginata and urticaria of nonserum origin are not infrequent on any large scarlet-fever service. These also are looked on as antigen-antibody reactions.

Scarlet fever has long been considered to be the origin of endocarditis. The toxin of scarlet fever does not in itself do any permanent damage to the endocardium, and only in the severest toxic cases is the myocardium injured in the eruptive phase. Scarlet fever, however, as a streptococcal disease may initiate rheumatic fever, and that is where the greatest danger of scarlet fever lies today. The exact relation of streptococcosis to rheumatic fever is not yet known, but that streptococcal infections do usher in rheumatic fever is beyond all doubt. It was formerly thought that transient murmurs in the course of scarlet fever were of no significance.[31] Subsequent experience has shown that the "will-of-the-wisp" murmurs that appear in the second and third weeks are sometimes followed within four weeks by electrocardiographic indications of myocardial damage, marked murmurs and even sudden cardiac failure. These are clearly of rheumatic-fever origin. It is of great interest that rheumatic fever is apparently likelier to occur after the more superficial infections than after the deeper invasions of the streptococcus, such as erysipelas and abscess formation. Ordinary scarlet fever itself is one of the most superficial of streptococcal infections, and as such it is a dangerous instigator of that far more serious condition, rheumatic fever. It is in this capacity that scarlet fever may lead to subsequent damage of the heart valves. Furthermore, streptococcal tonsillitis is just as dangerous. Thus, the superficial infective process rather than the rash appears to be the factor involved in the instigation of rheumatic fever.

Etiology

The concept of the etiology of scarlet fever has undergone remarkable changes in the last twenty years. In 1924, the Dicks[32] incriminated a hemolytic streptococcus as the causative agent in this disease. Their work helped greatly to clear the confusion concerning the specific agent, but their term "*Streptococcus scarlatinae*," tended to oversimplify the situation. With the development of methods for the identification of streptococci according to their antigenic make-up and for the classification of these organisms according to the group-specific carbohydrate material[33] and type-specific nucleoproteins,[34] a more exact basis was furnished for the study of the organisms that produce scarlet fever. It is now known that strains that produce scarlet fever in some patients may produce only localized diseases without a rash in others, and that in various communities of a single country and in the various countries of the world the streptococci that are responsible for the production of scarlet fever may be widely different antigenically, varying to a slight degree in the type of group-specific carbohydrate that they contain, and in a much greater degree in the nature of the type-specific nucleoprotein.

All the strains of streptococcus isolated from the pharynges or other local sites of infection in cases of scarlet fever belong to the group that produce beta hemolysis, as seen by a fairly wide, well-defined, clear ring of hemolysis about the colony in blood agar media. Analyses according to the method of Lancefield[33] of many strains for the type of carbohydrate that they contain have shown that more than 95 per cent of them belong to her Group A, the group that is known to be by far the most prevalent in all infections in man due

to the streptococcus. Although this evidence points to a marked degree of similarity between all the strains of streptococci that are isolated from cases of scarlet fever, such a generalization cannot be made because other groups — namely, Group C[35] and Group G[36] — have been shown to be capable of producing the same disease, although much less frequently than Group A. In any event, as pointed out by Brown and Schaub,[37] *Str. scarlatinae* is not *the* streptococcus of scarlet fever but is *a* streptococcus of scarlet fever.

That no single strain of streptococcus is involved in the production of scarlet fever becomes evident from the studies on the type-specific nucleoprotein present in strains of streptococci isolated from different cases of scarlet fever. Keefer and his coworkers[38] analyzed the literature on the distribution of the various types of streptococci demonstrable in scarlet fever and studied a large group of cases that came under their own observation. Their conclusions were that scarlet fever may be due to a wide variety of streptococci and that the predominant types may vary from one country to another, from one community to another and from year to year in the same community or country. They found that during an epidemic in a community one type predominated or two types thrived simultaneously. They also found that as an epidemic of scarlet fever progressed in the same community, one type might be replaced later by another. This same fact was noted last year at the Haynes Memorial Hospital, where early in the winter the predominant type isolated from cases of scarlet fever was Type 1, although there were six or seven other types present. In the spring and late winter, Type 2 became the organism most frequently isolated, although some cases were still due to Type 1. Keefer and his coworkers also point out that in sporadic cases of scarlet fever the infections are oftener due to types that are not concerned with the types causing the epidemic and that there is often wide scattering. They also state that the same type of streptococcus may be responsible for different clinical diseases — that is, scarlet fever in one person, tonsillitis in another, erysipelas in a third and rhinitis in a fourth — and that when one or more types are predominant in any community, the same types are usually isolated from persons with various diseases, such as scarlet fever, tonsillitis and otitis media. All the studies made during the last two or three years in various Army and Navy establishments have borne out the same fact; in other words, in any large group of men scarlet fever may be due to a number of different types of streptococci, although one or two types are usually found to predominate.[39]

In view of the ample evidence that the causative streptococcus in scarlet fever may differ not only in the type of group-specific carbohydrate content but much more frequently in the qualitative character of the nucleoprotein content, it appears that the concept of a single species of organism is hardly tenable. Furthermore, the facts that the organisms isolated from cases of scarlet fever are beta hemolytic streptococci and that they all produce erythrogenic toxin and may show similar fermentative ability do not constitute enough evidence to class them as a single species, particularly since it has been well demonstrated that they differ antigenically. The situation is somewhat analogous to that which prevails in the Salmonella groups. Although several strains of this group may cause acute enteritis, may be isolated from the feces of various persons and may show closely similar activity against carbohydrates, they are not classified as a single species. Indeed, they are separated into different species on the basis of an analysis of their antigenic constituents. Whereas in the case of the Salmonella group a specific name is usually attached to organisms that resemble each other closely in their invasive, cultural and biochemical activities, such a procedure is not necessary with the Group A beta-hemolytic streptococci, the type number sufficing to identify the organism.

The whole problem of the etiology of scarlet fever is further confused in view of the fact that organisms other than beta-hemolytic streptococci at times produce the same symptom picture. Many drugs, notably quinine and the sulfonamides, are capable of producing fever and a typical scarlatiniform rash in hypersensitive persons, followed by desquamation. These drug rashes, however, do not represent the invasion by an infectious agent, and therefore do not come under the concept of scarlet fever. Moreover, Aranow and Wood[3] point out that some strains of hemolytic *Staph. aureus* are capable of producing a symptom picture that is indistinguishable from clinical scarlet fever. They have described a case of osteomyelitis due to a strain of *Staph. aureus* in which a diagnosis of scarlet fever was made on admission to the hospital because the patient had a typical scarlatiniform rash and a raspberry tongue. No beta-hemolytic streptococci were cultured from the pharynx, but convalescent scarlet-fever antiserum was administered, and this blanched the rash. It was subsequently shown that the patient had osteomyelitis of the femur due to a strain of *Staph. aureus*, which was also cultured from the blood stream. Later the patient showed desquamation typical of the convalescent stage of scarlet fever. A study of this staphylococcus showed that it produced erythrogenic toxin that was closely related immunologically to that produced by some strains of beta-hemolytic streptococci. That this is not an isolated case of production of erythrogenic toxin by *Staph. aureus* is shown by a review of the literature on this subject by these same authors, who cite several other investigators who have demonstrated the production of a rash-producing toxin by a staphylococcus; at least forty-three strains that produce this

type of toxin have been described. Some of them have been isolated from other cases of osteomyelitis, and one from the pharynx of a patient who had a typical case of scarlet fever. Thus the present concept of the etiology of scarlet fever extends even beyond the Group A streptococci.

A rational concept of the etiology of scarlet fever appears to be one in which it is recognized that with certain rare exceptions the beta-hemolytic streptococcus is the agent that is responsible for the disease. It is usually Group A organisms that are responsible, however, and practically any of the forty-six serologic types of the Group A streptococci that have been identified may produce the disease, the predominant type varying with the year, the community and the country.

All the serologic types of the Group A streptococcus that produce scarlet fever have one property in common, namely, the ability to produce erythrogenic toxin. Lack of this toxin renders a strain incapable of producing the rash that characterizes scarlet fever. The mere ability of a strain to produce this toxin, however, does not necessarily imply that it will produce scarlet fever in every person in whom it gains a foothold. The immune state of the infected subject against erythrogenic toxin is the other determining factor. Thus, people who have an antitoxic immunity (Dick negative) develop only localized streptococcal infections without a rash, whereas those who have no antitoxic immunity (Dick positive) develop a scarlatinal rash. It is a well-established fact that many of the strains that produce only localized infections without a rash in certain people are capable in the test tube of producing erythrogenic toxin, which when injected into a susceptible (Dick-positive) person produces the typical reaction. Thus, a Dick-negative person with a localized infection — without a rash — can infect a Dick-positive one, who breaks out with scarlet fever.

Although the production of erythrogenic toxin appears on the surface to be the one property that all the streptococci that produce scarlet fever have in common, this point requires qualification, since it has been shown by Hooker and Follensby[2] that strains of streptococcus isolated from scarlet fever may produce two different types of erythrogenic toxin, A and B, which are distinguishable on the basis of both chemical and immunologic criteria. They have shown that a single toxigenic strain may produce A or B, both or neither. This difference in the quality of erythrogenic toxin produced by different strains of the hemolytic streptococcus is thought by these authors to be a possible explanation of the fact that some persons do not become Dick negative after having had scarlet fever, and that occasionally even the early administration of potent antitoxin does not appear to alter the toxic manifestations of scarlet fever in certain patients.

(To be concluded)

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CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

Weekly Clinicopathological Exercises

FOUNDED BY RICHARD C. CABOT

TRACY B. MALLORY, M.D., *Editor**

BENJAMIN CASTLEMAN, M.D., *Acting Editor*

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CASE 31181

PRESENTATION OF CASE

A fifty-six-year-old railroad fireman was admitted to the hospital because of malaise, weight loss and cough.

About twenty months before admission he had lobar pneumonia, which kept him in bed for two weeks. After three months he had recovered and felt quite well, but he never regained his full strength or weight. He returned to work despite the fact that his physician told him there was something wrong with his left chest. Eleven months before admission he suddenly developed malaise, loss of energy and ambition. At that time he was found to have a systolic blood pressure of "about 80" and he was given some medicine. After three weeks he improved and returned to work. He remained active until two months before admission, when he again experienced acute malaise and weakness of a severer nature than that of nine months before. This lasted three weeks. A third attack of weakness and malaise began one month before admission, and the patient remained in bed most of the time for about two weeks. Ever since the episode of pneumonia he had had a chronic "residual" cough which, especially in the morning after arising, was productive of white and gray-brown sputum, which was not malodorous and contained no blood. The cough was worse when he lay on his left side. One week before admission he began to wheeze on exertion. During the two years before admission he had lost about 35 pounds.

In the past he had had mumps, influenza and an infrequent cold. He was a moderate smoker and drank only an occasional glass of beer. His mother had died of diabetes, and his father of "locomotor ataxia." His wife and three children were living and well.

Physical examination revealed a rather emaciated man with an occasional loose cough. The heart sounds were faint and regular. There was slightly limited chest expansion on the left. The percussion note over the left base, both anteriorly and posteriorly, was duller than that on the right. There were diminished to absent tactile fremitus, breath sounds and voice sounds in the left chest anteriorly and posteriorly, extending from the mid-upper chest to the base. Expiratory wheezes were heard over the entire chest. The throat was normal. Abdominal and rectal examinations were negative.

The temperature, pulse and respirations were normal. The blood pressure was 100 systolic, 74 diastolic.

Examination of the blood showed a red-cell count of 5,250,000, with 15.1 gm. of hemoglobin, and a white-cell count of 12,300, with 85 per cent neutrophils. The serum protein was 6.6 gm. per 100 cc., and the nonprotein nitrogen 21 mg. A Hinton test was negative. An x-ray examination of the chest revealed the left hilus to be markedly increased in size. There was an area of increased density extending downward and posteriorly into the lower lobe. Within this area several rounded masses were seen (Fig. 1). The motion of the diaphragm was paradoxical, and the mediastinum shifted to the left on inspiration. The right lung appeared normal.

On the second hospital day a bronchoscopy was performed.

DIFFERENTIAL DIAGNOSIS

DR. GEORGE W. HOLMES: This story and the x-rays are typical of bronchial obstruction. In fact I think at the start that we can assume that this condition existed, and we must try to analyze the causes of bronchial obstruction. The most frequent cause is bronchiectasis, the next is tuberculosis, and the third, some form of primary neoplasm. In addition, bronchial obstruction is sometimes due to metastatic tumor and also to a foreign body. Let us go over the history with these things in mind and see if it will help us to arrive at a diagnosis.

This man was fifty-six years old, so that he was in the cancer bracket. He worked as a fireman and presumably was exposed to coal dust and other irritants. In the history it says that he was a smoker, but it does not say what he smoked: I think it makes a difference if one smokes cigarettes or a pipe. The record states that he had pneumonia and went to bed, but we are not told any of the details; in fact, we do not know whether he really had pneumonia. A story of pneumonia often follows bronchial obstruction. It really does not help us much in determining the cause of the obstruction. Carcinoma of the bronchus is frequently preceded by a story of pneumonia, and so are various other causes of bronchial obstruction. Following this episode he never regained his former weight, and the doctor warned him that he had something wrong with his chest. One wonders why the doctor did not go farther and find out what he had in the chest. Perhaps he did try, or the patient may have refused further study. Two years had passed before he came into the hospital, and all that time it was known that he had something wrong with his chest. I think that we have reached a period in chest examinations when

*On leave of absence.

it should be considered negligence not to take x-ray films of the chest in a man with a story such as this, just as it is considered negligence to make a negative diagnosis of fracture following injury without x-ray examination.

In carcinoma of the bronchus by far the most frequent early signs are pain in the chest, blood spitting and cough; the cough is usually brassy and unproductive. This man did not have pain in the chest and did not have any blood spitting, and the cough was said to be loose. All these facts are against carcinoma of the bronchus and in favor of some other cause, possibly tuberculosis.

The story was always one of weakness and malaise, which are not unusual complaints for carcinoma of the bronchus. In the family history they mention locomotor ataxia. He was married and had three children and later on had a negative Hinton test. I do not think we have to pay much attention to syphilis.

What do we get out of the physical examination? He was emaciated and had a cough and limited chest expansion. The statement about the percussion note is interesting. I wonder whether that interpretation would be a help or a hindrance to a clinical man. What do you think about it, Dr. Cass?

DR. JOHN W. CASS: I should say that it was simply a sign of bronchial obstruction.

DR. HOLMES: He had an expiratory wheeze. That is the most significant of all the physical findings. Expiratory wheeze means bronchial obstruction. He had no pain, and no elevation of temperature, pulse or respiration. It seems unlikely that he could have had an extensive suppurative process in the lungs. He had a low blood pressure on two occasions. I shall come back to that later.

The examination of the blood — the hemoglobin level, white-cell count and so forth — was within the limits of normal.

It is surprising how little the anteroposterior film (Fig. 1) shows, and one wonders why there were such definite signs on percussion over the left side of the chest. If you look at the lateral view you can see there is dullness in the posterior part of the chest in the region of the lower lobe. Then, if you look carefully at the left lung in the anteroposterior film you notice that it is brighter than the other side and that the number of markings in the lung are less than those on the other side. In other words, there is evidence of collapse of the left lower lobe. The lobe is hidden behind the heart shadow. It would he helpful if films had been taken with more exposure and, perhaps, a grid film, to determine the outline of the diaphragm. If the lung is collapsed the outline of the diaphragm is obscured. It would also be helpful to know if there were any cavities in that portion of the lung. In addition, there are these massive shadows at the left lung root. They are probably due to enlarged hilar nodes and are of considerable importance in the diagnosis and also when considering the question of operability. There is no evidence of calcium deposits in any of these nodes, and no calcified masses anywhere in the lung, which is some evidence against tuberculosis.

In a patient with a long story of bronchial obstruction one always has to think of an adenoma, a benign tumor of the bronchus, but this patient's

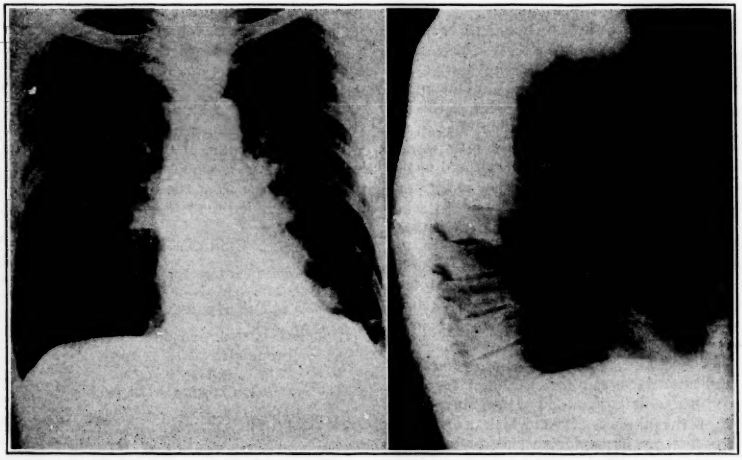

FIGURE 1. *Anteroposterior (left) and Lateral (right) Roentgenograms of Chest.*

history was only two years in duration, which is about the duration for a carcinoma of the bronchus. Adenoma of the bronchus is usually associated with a certain amount of infection, and the patients have a story of raising foul sputum. I cannot rule out an adenoma of the bronchus, but I doubt that that is what he had. I do not believe that we have to consider bronchiectasis seriously, for the same reasons.

Metastatic malignant tumors, including lymphoma, have to be considered. We have had only one case that I know of in which the mass produced compression of the bronchus. As a rule, metastatic tumors of the lung do not cause bronchial obstruction.

That brings it down to whether or not this patient had primary carcinoma of the bronchus or tuberculosis, or both. There is another point in the history that we must consider at this time, that is, the low blood pressure and the story of malaise and weakness coming on at varying intervals and finally bringing him into the hospital. This low blood pressure and a process in the lungs that could be either tuberculosis or cancer makes one think of something involving the adrenal glands. Metastases to the adrenal glands are not rare in carcinoma of the lung, and their involvement is quite frequent in tuberculosis. So again we are left in the same predicament. We do not know whether this was carcinoma or tuberculosis.

Then in the x-ray examination there is something quite significant. He is said to have had paradoxical movement of the diaphragm. That probably means paralysis of the phrenic nerve, which is a fairly frequent occurrence in carcinoma and rarely occurs in other conditions. We have one case reported in which there was paralysis of the phrenic nerve with tuberculosis. This, of course, may be that case. I should like to be absolutely certain about that statement. One would think it would be a hard thing to miss, but it is not. The whole differential diagnosis, or a large part of it, so far as I am concerned, depends on that single statement.

Was any sputum obtained?

DR. BENJAMIN CASTLEMAN: No sputum examination is recorded.

DR. HOLMES: It is an important examination, and we might be able to clinch the diagnosis between carcinoma and tuberculosis on that alone.

Leaving aside the fact that cases selected for this conference are sometimes rare, one must arrive at the conclusion that this patient had a primary carcinoma of the bronchus, with changes of some sort in the adrenal glands.

The record stops at the bronchoscopy. I should like to go a step farther and speculate what should have been done about this man. In the first place I think that he should have had x-ray films taken within a month after the first episode and that he should have had a bronchoscopic examination at about the same time. On the data that we have here he was inoperable so far as cure is concerned, because the nodes were involved. There is evidence that he may also have had involvement of the adrenal glands. Should he have had x-ray treatment? We do it usually to relieve symptoms rather than with the hope of cure. This man had no symptoms that could be alleviated by radiation.

DR. RICHARD H. SWEET: In regard to the paradoxical motion of the diaphragm, do you think that that would be the case if there had been no evidence on the x-ray films? I assume that the diaphragm was not high on the left side. In other words, I am inclined to doubt the observation.

DR. HOLMES: The fact that the diaphragm was not high makes you doubt the observation?

DR. SWEET: Yes.

DR. EDWARD B. BENEDICT: Bronchoscopy showed the carina to be normal, sharp and not fixed. At a point in the region of the orifice of the left upper lobe there was a smooth, pearly-gray mass, apparently arising from the upper lobe orifice, which partially occluded the left main bronchus.

CLINICAL DIAGNOSIS

Carcinoma of lung.

DR. HOLMES'S DIAGNOSIS

Bronchial obstruction due to primary carcinoma of bronchus.

ANATOMICAL DIAGNOSIS

Bronchial adenoma.

PATHOLOGICAL DISCUSSION

DR. CASTLEMAN: The biopsy specimen showed that this was a benign bronchial adenoma. Dr. Benedict followed the man from then on.

DR. BENEDICT: I followed him at frequent intervals. He had eleven bronchoscopies over a five-year period, and on almost every occasion I found some tumor tissue, which I removed and sent to the laboratory, with a report of "benign adenoma" each time. In the meantime, the bronchial obstruction was relieved and he was symptomatically entirely well, except for a slight morning cough. He gained weight and returned to full-time work. We thought that behind the adenoma in the left lower lobe there were collapse and bronchiectasis. In a younger man we would have advised lobectomy, but in this patient, because of his age and the absence of symptoms, we went ahead observing him bronchoscopically. Each time there was evidence of adenoma. Because of the possibility that there was an extrabronchial adenoma and because some of the cases reported in the literature occasionally show a low-grade malignancy I finally put it up to the patient to have a lobectomy to get rid of the tumor and the irreparably damaged left lower lobe.

After several months' consideration he re-entered the hospital for lobectomy.

DR. SWEET: There are one or two interesting things about this case. I do not believe that he ever had paradoxical motion of the diaphragm, because it seemed to be quite active at operation. Another point is that the x-ray picture shows the anatomic findings with great accuracy. The aerated lung on the left side was the upper lobe, which filled the left chest except for one large bleb about the size of the palm of my hand. Because of Dr. Benedict's bronchoscopic findings, and having been told that the lesion was close to the upper lobe, I was extremely careful in doing the operation to search for tumor and was prepared to do a pneumonectomy. Dr. Evarts Graham* says that it should be done on all these patients, but in this clinic we believe that a lobectomy, if it includes the entire tumor, is sufficient. He believes that the histology indicates that such tumors are potentially malignant.

DR. BENEDICT: All bronchoscopies except the first one showed that most of the tumor was in the lower lobe.

DR. SWEET: Because of that, after removing the lobe, I looked down the bronchus, with the bronchus open, and made sure that there was no tumor.

DR. CASTLEMAN: The shrunken left lower lobe that we received showed no evidence of tumor. In other words all the tumor had been removed bronchoscopically, so far as we could tell grossly, and this was confirmed microscopically. The lobe, however, was irreparably diseased with extensive bronchiectasis. The upper half of the lobe was collapsed, and there was beginning fibrosis. In the lower portion there were cystic bronchiectatic cavities measuring up to 2.5 cm. in diameter. I think that it is a good idea to remove these diseased lobes, even though some of the patients have no symptoms. We had one young boy who had an adenoma almost completely removed. He had ten to fifteen bronchoscopies over a period of years, but the left lung remained collapsed. He went into the Army and died about seven or eight months later of amyloid disease, owing to the chronic infection that he continued to have in the obstructed lobe. It is also interesting that this boy had a metastasis in a bronchial lymph node.

I agree with Dr. Sweet that the large majority of these patients do not develop a malignant tumor, but we have had 2 cases in our series of about 40 in which the regional nodes were involved with the same benign-like adenoma as the primary tumor; in neither of these cases had it gone beyond the regional nodes. Such tumors act much like the argentaffin or carcinoid tumors in the small intestine, which occasionally have metastases to the regional nodes, producing small-bowel obstruction, but rarely go any farther. The metastasis may remain in that location for years.

DR. HOLMES: The bronchiectasis followed the adenoma?

DR. CASTLEMAN: Yes; it was a secondary bronchiectasis.

DR. HOLMES: Is it not unusual for patients with this type of tumor to be so free from fever?

DR SWEET: The patient did have several attacks of so-called "pneumonia."

DR. HOLMES: The X-ray Department failed me by observing paradoxical motion of the diaphragm, which he did not have.

DR. SWEET: I tried to point that out.

DR. BENEDICT: Dr. Edward Churchill always believed that this patient had carcinoma, and even after bronchoscopy and after the pathological report of adenoma came back, he was still inclined to believe that it was a carcinoma because of the x-ray findings.

DR. HOLMES: That one statement of paradoxical motion would also have prevented me from accepting the diagnosis of adenoma.

DR. CASTLEMAN: The enlarged hilar lymph nodes were undoubtedly inflammatory. There are later x-ray films that do not show them.

DR. SWEET: At operation they were not particularly significant.

DR. BENEDICT: The sharp, freely movable carina by bronchoscopy at the end of two years was against carcinoma.

DR. CASTLEMAN: The patient probably did not have as much bronchiectasis at the time the first films were taken as was found in the resected lobe; it must have increased during the six years that intervened.

*Graham, E. A. Problem of so-called adenoma of bronchus. An unpublished paper presented at a meeting of the American Association for Thoracic Surgery, Chicago, May 5, 1944.

CASE 31182

PRESENTATION OF CASE

First admission. A twenty-four-year-old man was admitted to the hospital complaining of massive hemoptysis.

At eight years of age the patient was acutely ill with joint pains, severe chest pain, fever and malaise. A few weeks later he developed chorea. He was maintained on strict bed rest in a hospital for ten months. He had prolonged elevation of pulse and temperature, but improved gradually with rest and routine care. He was seen in the Cardiac Clinic of the Out Patient Department at the age of twenty years. He worked every day with a prescribed half hour of rest every noon and had no complaints. A physical examination at that time revealed a fairly muscular young man, with a slightly sallow complexion. The lungs were clear. The heart was enlarged, the apex beat being 9 cm. from the midline in the left fifth interspace. A systolic thrill was readily felt to the right of the sternum in the

second interspace. An apical systolic and a mitral diastolic rumble without a presystolic phase were present. A loud, rough aortic systolic murmur associated with a thrill and an aortic diastolic murmur were heard. The pulmonic second sound was prominent, and the aortic was absent. The blood Hinton and Wassermann tests were negative. The temperature and respirations were normal. The blood pressure was 100 systolic, 60 to 40 diastolic. There was a Corrigan pulse, with a regular rate of 80. During the following three years he made frequent visits to the Out Patient Department and showed little change. The heart size increased slightly, and his blood pressure was 118 systolic, 70 diastolic. He was able to climb two or three flights of stairs without dyspnea, and managed to work regularly at jobs that did not require much physical exertion. He smoked one package of cigarettes daily. He came to the Medical Clinic of the Out Patient Department at the age of twenty-three, complaining of expectoration of bright-red blood, unmixed with sputum. The first attack had occurred suddenly at 10:30 p.m., three days before admission. There were no premonitory symptoms, and he had not been exerting himself unduly at the time. The attack consisted of a choking sensation without dyspnea, followed by coughing and expectoration of about one cupful of blood. This episode lasted five minutes, then ceased abruptly, and was not associated with any discomfort. During the month preceding these episodes he had changed from automobile driving to pick-and-shovel work because he could find no other job. Physical examination at that time revealed a slight increase in the size of the heart, which extended by percussion to 11 cm. from the midsternum in the left fifth interspace. A snapping first sound at the apex, which occasionally became weak and muffled without respiratory relation, was preceded by a crescendo presystolic murmur. The systolic murmur at the apex was unchanged. No second sound was heard at the apex or over the aortic area. The diastolic murmur over the aortic area and to the left of the sternum lasted throughout diastole. A loud and extremely rough systolic murmur was present to the right of the sternum in the second, third and fourth interspaces. The rate was regular at 64, except for an occasional premature systole. The blood pressure was 108 systolic, 60 diastolic. The lungs were normal, and there was no clubbing of the fingers, and no edema. The liver could not be felt, and the neck veins were not distended. Careful examination revealed no evidence of oral or nasopharyngeal bleeding. The red-cell count was 3,800,000, and the hemoglobin 55 per cent. X-ray examination of the chest revealed enlargement of the heart in the region of the left ventricle. The aorta and lungs were normal. An electrocardiogram showed a tendency to right-axis deviation; the PR interval was 0.19 second, and T_3 was inverted. Following this examination the patient did not come to the Out Patient Department for nearly two years when, at the age of twenty-five, he returned. He had had hemoptysis almost every day for the preceding week, producing from half to one cupful of bright-red blood on each occasion. He had a "chest cold," felt weak and experienced dyspnea on exertion and slight orthopnea. He was quite pale, with a red-cell count of 3,980,000 and a hemoglobin of 8.2 gm. A few crackling rales were heard over the lungs anteriorly and posteriorly. Physical examination of the heart was as last described. The blood pressure was 95 systolic, 50 diastolic. There was no venous distention, clubbing of the fingers or enlargement of the liver. There was slight pitting edema of the lower portion of the legs. He was digitalized and given Mercupurin and ferrous sulfate. Fluids and salt were restricted. On this regime the patient improved and returned to work. Seven months later the patient was admitted to the hospital because of recurrent hemoptysis. On the preceding day he had contracted an upper respiratory infection and on the day of admission had had repeated coughing spells with hemoptysis. Shortly before admission he had a shaking chill.

On examination the patient was slightly cyanotic, dyspneic and orthopneic and had slight neck-vein distention. The heart revealed no change. There were crepitant rales over the upper lobes, and loud gurgling rales over both bases anteriorly and posteriorly. The liver could be felt at the costal margin and was slightly tender.

The temperature was 103.6°F., the pulse 84 and the respirations 36.

X-ray examination of the chest revealed a homogeneous area of increased density occupying the middle third of both lung fields from the hila to the lateral chest walls; there was no evidence of fluid (Fig. 1). An electrocardiogram revealed a PR interval of 0.20 second; P_1 and P_2 were prominent, and the axis was borderline. He responded well to intravenous sulfadiazine, as well as Cedilanid and an oxygen tent. The temperature was normal on the third day, and the chest cleared promptly. Several sputum and blood cultures were negative. He was discharged on the tenth hospital day.

Second admission (two months later). Two weeks after discharge he returned to the Out Patient Department after several episodes of hemoptysis, and because he had a "chest cold." The cough was worse when he was recumbent. There was no evidence of right heart failure. More rest, continued digitalis and diuretics were prescribed. He was subsequently readmitted to the hospital because of hemoptysis that was severer than any previous attack of bleeding. This episode again followed the onset of a "cold" and produced "almost a quart of blood."

During a two-month interval in the hospital he was afebrile after the third day. During the first

week the sedimentation rate was 0.7 mm. per minute and the PR interval was 0.22 second. He had frequent severe hemoptyses. His white-cell count varied from 5000 to 12,000, and the red-cell count rose from 3,600,000 to 5,300,000 on discharge. The urine was negative. The cardiothoracic ratio was 18.2:30.3. X-ray examination of the lungs was consistent with pulmonary edema or congestion. An electrocardiogram showed an inverted T_3 and a tall T_4, with a PR interval of 0.21 to 0.26 second. Physical examination of the heart was unchanged. The lungs cleared fairly well before discharge.

Third admission (ten months later). The patient developed another cold with sore throat and was readmitted to the hospital after several episodes in rapid succession of hemoptysis up to 200 cc. of bright-red blood. The ten-month interim had been almost entirely free from hemoptyses and the lungs had been clear. He had done no work, however.

The temperature was 104.2°F., the pulse 140, and the respirations 30. The blood pressure was 100 systolic, 34 diastolic.

The lungs presented a few fine rales, and the heart almost reached the anterior axillary line. During the first few hours after admission he coughed up 400 cc. of blood, and smaller amounts on the succeeding three days. The red-cell count fell to 3,800,000. Electrocardiographic and x-ray studies showed no heart or lung changes. The temperature, the pulse and the respirations returned to normal on the fourth hospital day, and a slow blood transfusion was given. He was discharged on the twentieth day, nearly two weeks after the last hemoptysis.

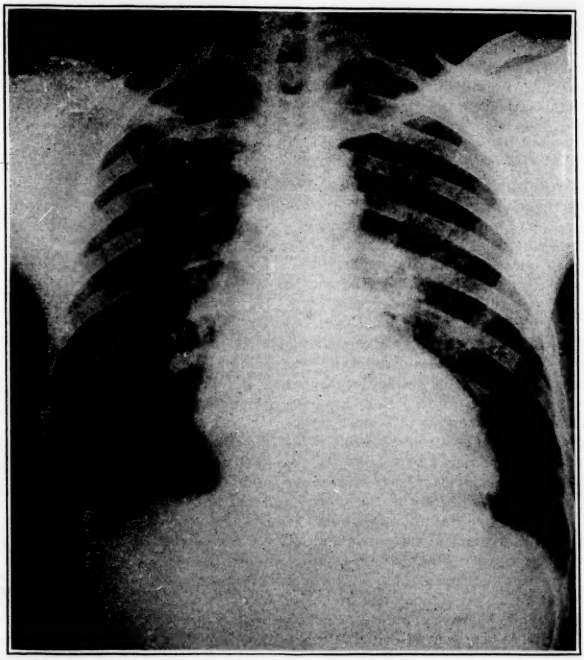

FIGURE 1. *Roentgenogram of Chest.*
Note the fine nodular density throughout the lung fields, as well as diffuse increased density centrally in each field.

Final admission (twelve months later). The patient worked for a few months as a painter, then went to Florida to rest. He returned by bus and, while en route to Boston, had the first hemoptysis that he had experienced in twelve months. On the day of admission he coughed up about 500 cc. of bright-red blood.

The temperature, pulse and respirations were normal. The blood pressure was 110 systolic, 50 diastolic.

An electrocardiogram showed partial auriculo-

ventricular block. The heart was unchanged except for further enlargement. The point of maximum impulse lay in the anterior axillary line in the fifth and sixth interspaces. The lungs were clear. The liver could not be felt, and there was no edema. Examinations of the urine and blood were negative. The serum nonprotein nitrogen and total protein values were normal. The sedimentation rate was normal. A cephalin flocculation test was ++++ in twenty-four hours.

The patient coughed up over 600 cc. of blood on all but one of the first five days after admission. After the fourth day, chest examination was difficult because of loud tracheal rales. Dyspnea and cyanosis progressed as the lung fields increased in dullness. On the sixth hospital day he expired quietly, at the age of twenty-seven years.

Differential Diagnosis

Dr. Edwin O. Wheeler: Obviously this man had severe, prolonged rheumatic fever at the age of eight years. There was no further evidence of rheumatic fever for a period of twelve years.

There is not much question concerning the type of heart disease that this patient had. At the age of twenty years he presented a typical picture of rheumatic heart disease, with mitral and aortic stenosis and insufficiency and cardiac enlargement. He had no evidence of congestive failure, and he had not developed auricular fibrillation. During the next three years the mitral murmur became crescendo in character and the first sound at the apex became snapping, suggesting that the mitral stenosis had increased in severity and was of the typical adult type. The aortic insufficiency was not of marked degree. The electrocardiogram at the same time revealed an axis that was within normal limits, suggesting that the two valvular lesions, aortic and mitral, tended to balance each other so that no ventricular preponderance developed.

It is rather unusual for a patient to cough up so much blood. It was pure blood, apparently, and he had repeated attacks. There was no evidence of pulmonary congestion during the first attack. I wonder how soon after the episode of hemoptysis that observation was made. In a patient who had bled that much one would expect to find rales at the lung bases. We do not know whether he had fever or leukocytosis during the early episodes, though such did occur later on. Evidently the physician doing the examination did not believe that the patient had a chest cold, since it is noted in quotation marks. Pulmonary congestion may well have produced this sensation, for he had had dyspnea on exertion and slight orthopnea. There was little or no evidence of right-sided heart failure during these early attacks except for the slight edema of the legs. There was, however, considerable evidence of pulmonary congestion at the time of his first admission for an episode of hemoptysis, with no evidence of pulmonary consolidation or of a localized pulmonary lesion.

May we see the x-ray films?

Dr. Laurence L. Robbins: In the first film, taken in the Out Patient Department, there is increased density in the central portion of each lung field. The heart is prominent in the region of the left ventricle, and there is left auricular enlargement. One film shows prominence of the pulmonary artery. I do not see any calcification in either the mediastinum or the aortic valve, but that cannot be determined without fluoroscopy.

This film was taken three years later. The heart has increased in size. In addition there is diffuse increased density centrally located and a fine nodularity scattered through the lung field. Another film taken a year later shows an increase in the nodularity but no change in the central shadow (Fig. 1).

Dr. Wheeler: Is the nodularity consistent with that which we see in long-standing mitral disease?

Dr. Robbins: It is consistent with pulmonary congestion and edema.

Dr. Wheeler: There is no evidence of primary pulmonary disease?

Dr. Robbins: Not that I can see.

Dr. Wheeler: The PR interval was still within normal limits. The prominent P_1 and P_2 are in keeping with auricular enlargement. Axis deviation had not developed, showing no preponderance of either ventricle; the patient probably had hypertrophy of both ventricles.

The story at the time of his first admission suggests that the diagnosis of pneumonia, which was entertained, was incorrect. He recovered rapidly and had no x-ray evidence of pulmonary consolidation, and the sputum and blood cultures were negative. A diagnosis of pneumonia is often made in patients with mitral stenosis who present these symptoms.

At the time of his second admission he again had hemoptysis, fever and some leukocytosis, which responded much more quickly to bed rest than is the case with pneumonia or pulmonary infarction.

What happened to the sedimentation rate during the second hospital admission?

Dr. Ronald C. Sniffen: Only one sedimentation rate is recorded.

Dr. Wheeler: Quite a degree of partial auriculoventricular block was present at times, suggesting active rheumatic fever. Again, there was no evidence of pulmonary disease as a cause for the hemoptysis.

The third hospital admission was a repetition of the two preceding entries, and the fourth admission varied only in that the patient failed to recover and apparently died in pulmonary congestion.

This, briefly, then was a patient with rheumatic heart disease with aortic and mitral involvement who suffered from recurrent massive hemoptyses

for a period of several years. Hemoptysis of this degree is not often seen in any sort of intrinsic pulmonary disease, such as tuberculosis, bronchiectasis or new growth. Hemoptysis may occur frequently in these conditions but is rarely so massive. Furthermore, since the lungs were apparently normal, we can rule out the above conditions. Certainly a neoplasm should have manifested itself after a period of four years. Pneumonia, which was suspected once in this patient, was evidently not present.

Other forms of heart disease will occasionally produce bloody sputum. Left ventricular failure due to either hypertension, aortic-valve disease or coronary disease may produce pulmonary congestion and blood-streaked or pinkish sputum but rarely, if ever, does it cause massive hemoptysis of pure blood. There are other less frequent causes of blood spitting, such as aortic aneurysm, which may rupture into a bronchus and produce a massive hemorrhage but does not cause repeated attacks over a period of four years. There is no evidence of aneurysm in the x-ray films.

One diagnosis that I believe should be seriously considered is pulmonary infarction. Pulmonary infarction frequently occurs in patients with rheumatic heart disease, owing either to pulmonary embolism or to pulmonary thrombosis. There is usually, however, only a small quantity of blood coughed up in this condition, and with repeated attacks one would expect to find good x-ray evidence of either recent infarction or scarring from old infarcts. He had none of these.

Patients with mitral stenosis occasionally cough up large amounts of pure blood. The mechanism involved is not that of heart failure, but rather that of an overstrong right ventricle, which tends to flood or overcrowd the pulmonary circulation with blood that has difficulty in getting out of the pulmonary bed because of the tight mitral stenosis. Such a mechanism may lead to pulmonary congestion, to pulmonary edema or, less frequently, to the rupture of small vessels with pulmonary hemorrhage, as observed in this case, a syndrome that has been called "pulmonary apoplexy." Patients with this syndrome do not necessarily have heart failure. This patient showed no evidence of right-sided heart failure in his early attacks of hemoptysis. In fact, the precipitating cause for his attacks is not clear. Hard work is said to precipitate pulmonary apoplexy and did precede his first attacks. Tachycardia is another cause, the rapid heart rate tending to flood the pulmonary circulation, but this was not present in this case. It is interesting to note that he continued to have normal rhythm. Most patients with this syndrome do. I believe, then, that the attacks of hemoptysis were due to the mechanism described above and not to left ventricular failure.

Finally, the question should be raised whether or not this man had active rheumatic fever. He did have partial auriculoventricular block from time to time, and the sedimentation rate was rapid on the one reading that we have. I therefore suspect that he had active rheumatic fever during his last two or three hospital entries.

Dr. Conger Williams: I agree with Dr. Wheeler's diagnosis. This man had an interesting collection of symptoms. The most mysterious thing about the syndrome of pulmonary apoplexy is the elevation of temperature, often seen but never adequately explained. We are following such cases in the Out Patient Department. I remember another case with pulmonary edema admitted to a hospital no less than six times with a diagnosis of lobar pneumonia. No one thought it odd that the man should have pneumonia so often and recover so rapidly, that is, within three or four days of the onset. It is often difficult in this disease to restrain the attending physician from giving sulfonamides.

Clinical Diagnosis

Rheumatic heart disease, with mitral and aortic stenosis and insufficiency.

Dr. Wheeler's Diagnoses

Rheumatic heart disease, with mitral and aortic stenosis and insufficiency.
Syndrome of "pulmonary apoplexy."
Active rheumatic fever and congestive failure.

Anatomical Diagnoses

Rheumatic heart disease.
Acute and chronic rheumatic endocarditis of the mitral, aortic and tricuspid valves.
Stenosis of the mitral and tricuspid valves.
Cardiac hypertrophy and auricular dilatation.
Chronic passive congestion of lungs and liver.

Pathological Discussion

Dr. Sniffen: At the time of death this man was extremely cyanotic. His heart weighed 550 gm., with pronounced auricular dilatation. The mitral valve was stenotic and just admitted the tip of one finger. The aortic valve was not stenotic, but the cusps were somewhat interadherent, thickened and partially calcified. Below the aortic orifice on the posterior ventricular wall, the endocardium was rippled, like the sand on a beach, and three small accessory cusps had formed, indicating aortic regurgitation. The tricuspid valve measured 9 cm. in circumference (normal, 14 cm.), and its leaflets were somewhat thickened. On one cusp of each of these three valves there was a small area of ulceration covered by a fine layer of platelet thrombus. The

largest ulcer was on the noncoronary aortic cusp and measured 1 cm. in diameter. Microscopic sections of these lesions showed an active valvulitis, with endothelial destruction. The character and disposition of the reacting cells within the leaflet were those of rheumatic valvulitis. No bacteria could be found in the overlying thrombus. The right ventricular wall was 9 mm. in thickness, which is about twice the normal size; the left ventricular wall measured 23 mm. There was no increase in pericardial fluid.

There was no pleural fluid, and no ascites. The liver, however, showed chronic passive congestion with a mild central necrosis. The lungs weighed almost 2000 gm. and were dark, firm and meaty. Microscopically they showed the changes that we associate with increased pressure in the pulmonary circulation. They were, however, not so pronounced as those described by Parker and Weiss* in cases of severe mitral stenosis. Each alveolar wall showed many more capillary cross-sections than normal, and these capillaries were often dilated to several times the diameter of a red cell. Furthermore, many alveolar walls showed pericapillary edema, which compressed the capillaries. I believe that this is important for, in mitral stenosis, if pericapillary edema is present, gaseous exchange must be hindered. At a later stage in the process some alveolar walls were thickened by a deposit of collagen fibrils, associated with capillary narrowing. In the alveolar spaces there was extensive recent and old hemorrhage, the evidence for the latter being the presence of large numbers of hemosiderin-laden phagocytes. Intra-alveolar edema was slight and scattered. The large and small pulmonary arteries showed sclerosis, but it was not severe.

No Aschoff bodies were found in the myocardium, but unfortunately not enough material was studied to answer the question whether an active myocarditis was present.

*Parker, F., Jr., and Weiss, S. Nature and significance of structural changes in lungs in mitral stenosis. *Am. J. Path.* 12:573-598, 1936.

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CLOSING THE RANKS

The continuance of a democracy is dependent on the ability of its people to hold to their ideals of liberty and to find and follow high leadership. This democracy of ours, changing as it has been with the changing years, undergoing modifications in the pattern of its progress, still youthful enough and great enough to be flexible, has always found great leaders in its periods of crisis.

Emerging safely now from the darkness of our second great national crisis, we have achieved unanimity in the realization of the loss of a great leader. So it was after the death of Lincoln. Constructive, positive leadership cannot exist without opposition, but under such able guidance as we have had, that opposition is loyal, co-operative and appreciative.

If there is one prayer that we can all join in now, it is that our new president, with the best wishes of his country, will have strength to lead it onward along the path of victory and peace. This he can have only with our full loyalty and support, as we had given them to his predecessor. We must close the ranks and push on.

PATULIN INEFFECTIVE AGAINST THE COMMON COLD

In November, 1943, a group of British investigators reported on a new antibiotic agent, patulin, obtained from a strain of *Penicillium patulin*.[1] This substance was considerably more toxic than penicillin but had a wider range of activity and seemed to be effective against gram-negative bacilli. In preliminary clinical trials in uncomplicated cases of the common cold among naval personnel, there appeared to be a definitely higher cure rate after forty-eight hours among persons who sniffed patulin or sprayed their noses and throats with the drug than there was in comparable controls who used a buffer solution without patulin.

These observations were of considerable interest, since common colds constitute a large source of man-days lost, particularly during the early period of training of fresh recruits and among large industries. Because of these favorable results, the director of pathology and the consulting physician to the British War Office sponsored a series of clinical and laboratory trials in military personnel. A report of their findings has recently been published.[2]

Patulin was found to be highly toxic for mice, more so when given subcutaneously or intraperitoneally than when injected intravenously. In rabbits, solutions of patulin, unless extremely dilute, produced local reactions in the cornea and in the skin. Small amounts, injected intra-allantoically, resulted in the death of chick embryos. In therapeutic experiments in mice it was shown to have no effect on influenza A virus infections. In mice treated with patulin and infected with typhoid

bacilli the death rate was higher during the first two days after injection than in controls that received no patulin. The controlled clinical trials in the treatment of the common cold with patulin showed no advantage from its use as compared with that of a buffer solution without patulin.

These investigators emphasized the great difficulty in assessing the effect of any treatment in the common cold in view of the lack of objective signs that can serve as a check on subjective findings. They attempted to eliminate bias during treatment and in evaluating the results. Neither the investigators nor the patients knew which type of treatment was used, patulin or control. All the results were obtained before an analysis was attempted. The main trial was carried out in 100 men at a recruiting establishment during the season of autumn colds. Half the men were treated with patulin, and the others with the control buffer solution that served as the vehicle for the patulin. The two groups were comparable with respect to age, severity of symptoms, duration before and after treatment and bacteriologic findings. No dramatic effects were observed in either group. The patulin solutions used in the final trials were tested for bacteriostatic action nine weeks after their preparation and after the trials had been concluded. They retained their full activity as compared with fresh solutions. It was concluded that patulin had no demonstrable effect on the course of this series of colds.

When the preliminary results of these trials became known, the British Medical Research Council undertook an extensive investigation of the contradictory claims.[3] A study was carried out in several large factories and post-office units widely scattered throughout England. In these studies the test solutions were used as nose drops, but in two additional trials in public schools the solutions were also used as sprays. These clinical trials were supervised by local medical officers, and all the subjects were volunteers. Attempts were made to exclude cases of hay fever and chronic respiratory disease, and a uniform record system was used. In order to reduce the chances of guessing, four different solutions were used in each establishment, two of them containing patulin and the other two containing only the buffer solution. The solutions were freshly made, being used before they were three days old. The results in 1348 cases were available for analysis. Of these, 668 were treated with patulin, and 680 with control solutions. No significant differences were found among the separate units. There was an almost identical percentage of cures at different intervals after the treatment was begun in those who received patulin and in those who were given the control buffer solution. This was true irrespective of the duration of illness at the time when treatment was started. From these studies, therefore, no evidence was found to indicate that patulin is effective in the treatment of the common cold.

It is only fair to point out that these results were obtained with a substance that is relatively toxic when applied locally. It may still be possible that other antibiotics that are both more effective against bacterial infections and more innocuous to tissues will prove helpful in the treatment of the common cold and educe the chances for the establishment of secondary bacterial complications. On the whole, however, most of the experiences thus far with the topical use of sulfonamides or penicillin in mixed infections have not been too encouraging.

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MASSACHUSETTS MEDICAL SOCIETY

The BLUE SHIELD

The Blue Shield, now in its twenty-seventh month of operation, is beginning to make significant gains. The plan has added more new members in the past six months than it acquired in the first eighteen months of business. With nearly 125,000 members and 4000 participating physicians, it would seem that the Blue Shield has established itself as a definite part of the medicoeconomic structure.

The Blue Shield is accomplishing two main objectives: with a service contract for low-income subscribers, it goes farther in the direction of complete protection for serious illness involving surgery than even federal planners and social economists have demanded; and because its policies are controlled and guided by the medical profession itself, the Blue Shield satisfies the demand by doctors that their profession not be dictated to by politicians or outsiders. The reconciliation of these two widely separated aims is difficult. The Blue Shield may properly take pride in the relatively small amount of friction and misunderstanding that have accompanied this complicated task.

Commercial insurance offers cash indemnity, which does not solve the problem for the low-income group. Federal plans, which may aim at satisfying the poor man's need, would accomplish this worthy end through methods that would, in the opinion of most physicians, stultify the medical profession with bureaucracy and federal regulations. The Blue Shield may well be the only possible solution that is tailored both to provide adequate protection for the low-income group and to maintain the highest standards of the medical profession, while preserving the physician's cherished philosophy of freedom of action. It must be reasonably expected that such an enterprise will have its quota of "headaches" and will involve a certain amount of what might be called "postoperative complications."

In 1943 and 1944, Blue Shield payments for surgery, assisting and aftercare, anesthesia and x-rays averaged about $70 per case. Advisory committees in the various specialties are making continuous progress toward reconciling the differences in opinions regarding certain fees, and in eliminating inconsistencies. Participating physicians are beginning to speak up for the plan, to recommend it for the consideration of influential employers with whom they are acquainted and to distribute Blue Shield pamphlets in their waiting rooms. An average of about five doctors are becoming participating physicians every working day. All this seems to forecast a bright future for the Blue Shield. But, as the December issue of *Fortune* opined in its article "United States Medicine in Transition," if the voluntary or noncompulsory way is to solve the nation's health problem, the following two conditions will have to be met: the country must be prosperous with reasonably full employment, so that people will be able to pay their own contributions without government help; and the government at all levels, employers, the great mass of potential patients and, above all, the medical profession must show a degree of social inventiveness and a determination hitherto unknown.

Physicians cannot afford to be perfectionists in this matter. The Blue Shield is a practical plan, in being, which is becoming more successful each day. It is in a better position now than ever before to make a new advance toward a better solution. The Blue Shield deserves the united support of the medical profession in Massachusetts.

DEATHS

BRAY — Thomas A. Bray, M.D., of Holliston, died April 1. He was in his fifty-first year.

Dr. Bray received his degree from Georgetown University School of Medicine, Washington, D. C., in 1922. He maintained a general practice in Worcester six years and then entered the New York Eye and Ear Infirmary, where he studied diseases of the eye, ear, nose and throat two years. He returned to Worcester, practicing his specialty and becoming a member of the staff of St. Vincent Hospital. He moved to Holliston in 1944 and was on the staffs of the Framingham Union and Milford hospitals.

His mother, his widow, three sons, one daughter, a brother and five sisters survive.

CASSELBERRY — Clarence M. Casselberry, M.D., of Newton, died February 23. He was in his seventieth year.

Dr. Casselberry received his degree from the University of Pennsylvania School of Medicine in 1897. He was a fellow of the American Medical Association.

His widow survives.

GOOKIN — Edward R. Gookin, M.D., of Washington, D. C., died recently. He was in his sixty-fourth year.

Dr. Gookin received his degree from Baltimore Medical College in 1906. He was a fellow of the American Medical Association.

A sister survives.

GREENE — Jeremiah A. Greene, M.D., of Cambridge, died April 6. He was in his sixtieth year.

Dr. Greene received his degree from Harvard Medical School in 1913. In 1938 he was appointed associate medical examiner for Middlesex County by Governor Charles F. Hurley. He was a member of the Cambridge Medical Improvement Society and the Massachusetts Medico-Legal Society.

His widow, two sons and two daughters survive.

HOPKINS — William T. Hopkins, M.D., of Lynn, died April 10. He was in his seventy-seventh year.

Dr. Hopkins received his degree from Boston University School of Medicine in 1890. He was formerly Lynn health commissioner. He retired seven years ago after practicing in Lynn for more than fifty years. He was a fellow of the American Medical Association.

His widow, four sons and a brother survive.

NOYES — Nathaniel K. Noyes, M.D., of Plymouth, died April 12. He was in his eighty-first year.

Dr. Noyes received his degree from Dartmouth Medical School in 1890. During World War I, he was a captain in the Army Medical Corps. He was health-board physician in Duxbury from 1895 to 1925. He served as visiting physician at the Jordan Hospital, Plymouth, from 1915 to 1925 and as associate medical examiner of the Third Plymouth District for three terms. He was a fellow of the American Medical Association and a member of the Massachusetts Association of Health Boards and the Massachusetts Medico-Legal Society.

His widow, a son, two daughters and a brother survive.

YOUNG — Edward L. Young, III, M.D., of Brookline, was killed in action on March 24 in Germany. He was in his thirty-first year.

Dr. Young received his degree from Harvard Medical School in 1941. He entered the service in May, 1943, after serving his internship at the Massachusetts General Hospital. He went overseas in March, 1944, and was serving at a field hospital in the front lines.

His father, his widow, a daughter and a sister survive.

MASSACHUSETTS DEPARTMENT OF PUBLIC HEALTH

COMMUNICABLE DISEASES IN MASSACHUSETTS FOR MARCH, 1945

Résumé

Diseases	March 1945	March 1944	Seven-Year Median
Anterior poliomyelitis	5	0	0
Chancroid	4	2	*
Chicken pox	1319	3360	1604
Diphtheria	22	25	14
Dog bite	783	733	733
Dysentery, bacillary	4	19	4
German measles	225	316	147
Gonorrhea	459	509	327
Granuloma inguinale	1	0	*
Lymphogranuloma venereum	2	5	*
Malaria	76	55	0
Measles	624	3546	3245
Meningitis, meningococcal	22	60	10
Meningitis, Pfeiffer-bacillus	1	4	2
Meningitis, pneumococcal	5	7	†
Meningitis, staphylococcal	0	0	†
Meningitis, streptococcal	0	1	†
Meningitis, other forms	0	0	†
Meningitis, undetermined	3	20	†
Mumps	2790	1575	1303
Pneumonia, lobar	257	424	516
Salmonella infections	8	12	5
Scarlet fever	1701	1906	1457
Syphilis	412	674	479
Tuberculosis, pulmonary	239	228	283
Tuberculosis, other forms	20	21	21
Typhoid fever	1	2	3
Undulant fever	5	3	3
Whooping cough	854	376	874

*Made reportable December, 1943.

†Pfeiffer-bacillus meningitis only other form reportable previous to 1941.

Comment

Anterior poliomyelitis thus far this year has been at the highest point since 1932. The March total exceeded any since 1928.

Diphtheria, although well above the seven-year median, is running lower this year than last.

Meningococcal meningitis during the first quarter of 1945 was consistently below that of the corresponding three months of the last two years.

None of the relatively large number of cases of malaria reported in March originated in Massachusetts. They occurred in returned members of the armed forces.

Geographical Distribution of Certain Diseases

Anterior poliomyelitis was reported from: Cambridge, 1; Framingham, 1; Longmeadow, 1; Lynn, 1; Springfield, 1; total, 5.

Diphtheria was reported from: Boston, 3; Cambridge, 2; Framingham, 1; Hanson, 1; Lowell, 4; New Bedford, 2; Reading, 1; Somerville, 2; Taunton, 3; Wareham, 1; Winchester, 1; Worcester, 1; total, 22.

Dysentery, amebic, was reported from: Waltham Regional Hospital, 1; total, 1.

Dysentery, bacillary, was reported from: Boston, 1; Lawrence, 2; Northampton, 1; total, 4.

Encephalitis, infectious, was reported from: Brockton, 1; Chelmsford, 1; Everett, 1; Lexington, 1; Medford, 1; Quincy, 1; Weymouth, 1; total, 7.

Malaria was reported from: Boston, 1; Camp Edwards, 43; Cushing General Hospital, 11; Haverhill, 1; Lynn, 1; Melrose, 1; Natick, 1; Northampton, 2; Norwood, 1; Waltham Regional Hospital, 11; Waltham, 1; Winchester, 1; Worcester, 1; total, 76.

Meningitis, meningococcal, was reported from: Beverly, 1; Boston, 6; Chelmsford, 1; Fall River, 2; Fitchburg, 1; Holyoke, 1; Milton, 1; New Bedford, 1; Newton, 1; Palmer, 1; Plymouth, 1; Quincy, 1; Southboro, 1; Worcester, 3; total, 22.

Meningitis, Pfeiffer-bacillus, was reported from: Milford, 1; total, 1.

Meningitis, pneumococcal, was reported from: Amherst, 1; Lawrence, 1; Springfield, 1; Worcester, 2; total, 5.

Meningitis, undetermined, was reported from: Brockton, 1; Cambridge, 1; Springfield, 1; total, 3.

Salmonella infections were reported from: Beverly, 1; Boston, 1; Lynn, 1; Natick, 3; Revere, 2; total, 8.

Septic sore throat was reported from: Amesbury, 1; Arlington, 1; Boston, 9; Cambridge, 1; Fall River, 1; Lowell, 1; Medford, 1; Newton, 1; Quincy, 1; Salem, 1; Williamstown, 2; total, 20.

Trichinosis was reported from: Boston, 1; Douglas, 1; Haverhill, 1; Millbury, 1; Norton, 1; West Stockbridge, 2; total, 7.

Typhoid fever was reported from: Adams, 1; total, 1.

Undulant fever was reported from: Brookfield, 1; Northampton, 1; Northbridge, 1; Raynham, 1; Worcester, 1; total, 5.

CONSULTATION CLINICS FOR CRIPPLED CHILDREN IN MASSACHUSETTS UNDER THE PROVISIONS OF THE SOCIAL SECURITY ACT

Clinic	Date	Clinic Consultant
Haverhill	May 2	William T. Green
Lowell	May 4	Albert H. Brewster
Salem	May 7	Paul W. Hugenberger
Brockton	May 10	George W. VanGorder
Springfield	May 16	Garry deN. Hough, Jr.
Worcester	May 18	John W. O'Meara
Pittsfield	May 21	Frank A. Slowick
Hyannis	May 22	Paul L. Norton
Fall River	May 28	Eugene A. McCarthy

MISCELLANY

NOTE

Dr. Derek Denny-Brown, professor of neurology at Harvard Medical School and director of the Neurological Unit, Boston City Hospital, has been granted leave of absence by these institutions. He is to return to active military duty as a brigadier general with the British forces in India and South East Asia. In 1941, after two years of service in the field, he was temporarily released by the British Army to carry on research and teaching in the United States. He plans to resume his work in Boston at some later date.

CORRESPONDENCE

SPRING TONIC

To the Editor: The following anecdote written by the aged Rev. George Allen for Joseph A. Denny, of Leicester, Massachusetts, has recently come into my possession.

The Austin Flint referred to may have been a connection or even an ancestor of the two famous Austin Flints.

A. E. P. Rockwell, M.D.

Shrewsbury, Massachusetts

* * *

Joseph A. Denny, Esq.

Dear Sir:

Herewith I send the promised trifle. I prepared it soon after I last saw you, but knowing that if used at all, other matter must take precedence, I lay it aside to meet other calls of immediate claim; and you know out of sight is out of mind for some, especially an old man.

If you cannot decipher or guess at my hieroglyphics, perhaps I can tell what I meant, should you deem it important enough to trouble yourself about it.

Respectfully yours,

George Allen

Jan. 16, '74

* *

In conversation with the late Doctor Austin Flint when he was not far from his ninetieth year, I remarked to him that he had passed almost the whole of his professional life in Leicester, and had had many and special opportunities for learning the ways and sentiments of all classes of the people. To these remarks the Doctor of course assented.

I next intimated with respectful deference my opinion that a patient's faith in the skill and practice of his physician is sometimes an effective auxiliary to mere medical treatment. To this he replied, "It is so in cases not a few."

Perceiving that the Doctor had no disrelish for my freedom, I ventured a step further, and said: "Doctor, there have been and still are among the people, especially among the more ignorant, many fanciful conceits and absurd notions received by time-honored tradition and confidently relied on as helps or hindrances to a cure, and sometimes a wise prescription might contradict the delusion and disturb, if not destroy the confidence of the patient in the skill of his physician. How far may such delusions be honestly humored? How would you avoid disturbing the patient's confidence?"

The wary Doctor was at no loss for a reply, and ignoring all metaphysics, proceeded at once to illustrate the matter by relating an occurrence in his own early practice. I wish I could tell you the story just as he told it to me, but you know how impossible it is to put on paper flexile tones and looks, so often the very soul of utterance. However, you knew the Doctor too long and intimately not to remember well the imposing gravity he could assume in relating what was either facetious or ridiculous. The story as nearly as I can give it in lonesome black and white is the following:

Not long after I began to practice medicine in Leicester, a well-meaning man, the owner of a few acres and little or nothing else, called on me early in the spring of the year for professional relief. He complained in a forlorn way that he was ailing and felt very weak. He said he was afraid he should not be able to do his spring work himself, and that he was too poor to hire anybody to do it for him. With a pitiful look he desired me to do something for him if possible, that he might get better and be able to go to work in season for planting time. After a little examination and a few inquiries I thought I saw his case, and directed him to get some wild cherrytree bark, make a decoction of it, and take so much, so often daily in spirit. He listened intently to all I said, and when I ended my prescription he said with a look of eager honesty, "Doctor, shall I strip the bark up or down?" the belief being then, and having long been common, that if the bark was stripped up from the tree, the medicine would work up, and if stripped down, it would work down. I replied, "In your complaint it is not of the least importance whatever whether you strip it up or down but don't you strip it sideways for the world!! If you do it will work right between your ribs!" The poor man cheered up, got better, did his spring work, and in due season gathered in the fruits of his own honest labor.

BOOK REVIEWS

Artificial Pneumothorax in Pulmonary Tuberculosis: Including its relationship to the broader aspects of collapse therapy. By T. N. Rafferty, M.D. With an introduction by Henry S. Willis, M.D. 8°, cloth, 192 pp., with 26 illustrations and 14 tables. New York: Grune and Stratton, 1944. $4.00.

Artificial pneumothorax therapy for pulmonary tuberculosis came during the early twenties of this century like a gust of fresh air on a hot and parched summer's afternoon. For years, the world had been waiting hopefully for that elusive cure of the then greatest killer of men — pulmonary tuberculosis. In a period of a few years, it was hailed as an important advance in the control and alleviation of tuberculosis, and the acceptance of this form of therapy was greatly hastened because of the simplicity of administering it and the fact that all sanatoriums could make full use of it. Gradually, however, complications began to ensue and late results came to plague both the physician and the patient, and recently certain authorities have criticized this method of treatment. Thus, many phthisiologists became bewildered and did not know where to turn. It is therefore doubly welcome that Dr. Rafferty has penned this book and has shown them a way out.

In the eighteen chapters, the author has included not only his own experiences but also those of all the major clinics in this country. He knows the limitations of pneumothorax therapy but also understands its benefits. First, he is of the opinion that primary thoracoplasty has its place and should not necessarily follow pneumothorax therapy. He deprecates the use of pneumothorax therapy in extensive disease, in apical cavities and in bronchial involvement. He urges a trial of pneumothorax therapy for a few weeks and its abandonment if unsuccessful, especially if many adhesions prevent relaxation of the lung and pneumonolysis is not feasible. He is particularly emphatic on early re-expansion in cases of empyema and bronchial fistula. His discussion on tension cavity is scholarly. He has purposely left out many of the small details, and he deals more with effective and ineffective pneumothorax therapy, pneumothorax with phrenic paralysis, the prevention of complications and so forth.

This small book is written in an excellent style and should be read by all interested in the treatment of pulmonary tuberculosis.

Secretory Mechanism of the Digestive Glands. By B. P. Babkin, M.D., D.Sc., LL.D. 8°, cloth, 900 pp., with 220 illustrations. New York: Paul B. Hoeber, Incorporated, 1944. $12.75.

Dr. Babkin, who is research professor of physiology at McGill University, Montreal, and former professor of physiology at the University of Odessa, has written a valuable, up-to-date account of the experimental analysis of the secretory function of the digestive glands in carnivorous animals and man. The author writes easily and clearly. Throughout the book he rightly emphasizes the fact that it is only by a combined morphologic and physiologic approach that an understanding of the complicated phenomena of bodily function can be reached. Theories are interestingly presented but merely to serve as guides to facilitate further progress of the problems discussed. The mechanisms involved in the regulation of the secretory activity of the principal digestive glands are critically reviewed historically and then brought to the point where the author indicates the problems that await further investigation. An excellent bibliography covers eighty-five pages, and there is a good index.

This book should serve well the clinician who is charged with the clear understanding and the investigation of the diseases of the secretory apparatus of the alimentary canal.

The Radiology of Bones and Joints. By James F. Brailsford. M.D., Ph.D., F.R.C.P., F.I.C.S. Third edition. 8°, cloth. 440 pp., with 404 illustrations. Baltimore: Williams and Wilkins Company, 1944. $12.00.

In the third edition of this important roentgenologic treatise the author has attempted to add new information and "bridge the gaps" that were present in previous editions. The four years of war have added to the author's difficulties in gathering data concerning patients. In spite of the difficulties, a most creditable volume has been published. The only adverse change is a poorer grade of paper and a more condensed arrangement of the printed material. The roentgenologic discussion is divided according to anatomic location. This is probably as good a division as can be made considering the varied nature of the lesions described, but it occasionally makes dull reading. The varied picture of injury and disease shown in x-ray films is fully described. There are many helpful sketches, as well as numerous excellent photographs of the roentgenograms. A number of excellent teaching outlines list the diseases that frequently produce a certain type of change in bones in the various areas of the body. There are many references to the recent orthopedic literature.

This book represents twenty-five years of experience of one of the leading roentgenologists of England, and it can be recommended as an authoritative treatise on the roentgenologic aspects of lesions of the bones and joints. It should prove to be a most helpful reference book for both roentgenologists and orthopedic surgeons.

BOOKS RECEIVED

The receipt of the following books is acknowledged, and this listing must be regarded as a sufficient return for the courtesy of the sender. Books that appear to be of particular interest will be reviewed as space permits. Additional information in regard to all listed books will be gladly furnished on request.

Foundations of Neuropsychiatry. By Stanley Cobb, M.D., Bullard Professor of Neuropathology, Harvard Medical School, and psychiatrist-in-chief, Massachusetts General Hospital. Third edition, revised and enlarged, of the work

formerly known as *A Preface to Nervous Disease*. 8°, cloth, 252 pp., with 13 illustrations and 3 tables. Baltimore: Williams and Wilkins Company, 1944. $2.50.

This standard text has been revised and enlarged to bring it into line with recent advances in the field of neuropsychiatry. New knowledge in the experimental field hitherto confined to animals has been tested on human beings with success. A new chapter has been added dealing with the psychopathologic concepts that are important in medicine.

The Trials and Triumphs of the Surgeon, and Other Literary Gems. By J. Chalmers Da Costa, M.D., LL.D. Edited by Frederick E. Keller, M.D. 8°, cloth, 401 pp. Philadelphia: Dorrance and Company, 1944. $5.00.

Here for the first time are collected the minor writings of Dr. Da Costa, who was the Samuel D. Gross Professor of Surgery at Jefferson Medical College of Philadelphia from 1910 to 1930. The papers are mostly historical.

Gynecological and Obstetrical Urology. By Houston S. Everett, M.D., associate professor of gynecology, Johns Hopkins University, associate in gynecology, University of Maryland, assistant visiting gynecologist and gynecologist in charge of the Cystoscopic Clinic, Johns Hopkins Hospital, and visiting gynecologist, Church Home and Hospital, Hospital for the Women of Maryland and Union Memorial Hospital. 8°, cloth, 517 pp., with 220 illustrations. Baltimore: Williams and Wilkins Company, 1944. $6.00.

This new textbook is based on the author's ten years' experience in the teaching of urology in the Department of Urology of Johns Hopkins University Medical School. It has been written for medical students, general practitioners, gynecologists and obstetricians, who may be confronted with urologic problems in their practice. Particular stress has been made on the general nature of urologic disease and its relation to general medicine and the other specialties.

A Textbook of Pathology: Pathologic anatomy in its relation to the causes, pathogenesis and clinical manifestations of disease. By Robert A. Moore, M.D., Edward Mallinckrodt Professor of Pathology, Washington University School of Medicine, Saint Louis. 8°, cloth, 1338 pp., with 513 illustrations. Philadelphia and London: W. B. Saunders Company, 1944. $10.00.

This new textbook on pathology embodies a new departure in classification in which degeneration is approached by disturbances of metabolism rather than by anatomic types. In the section on special pathology, diseases with similar causes have been grouped together; and those diseases of which the causes are unknown or obscure have been considered according to the organ or the system in which they occur. Bacterial diseases have been classified according to the portal through which the bacterium enters the body and the source of bacterium. The work is well printed on good paper with a good type and copiously illustrated. There are extensive citations to the literature scattered throughout the text.

Taber's Dictionary of Gynecology and Obstetrics. By Clarence W. Taber, with the collaboration of Mario A. Castallo, M.D., assistant professor of obstetrics, Jefferson Medical College, gynecologist to St. Mary's and St. Agnes hospitals and obstetrician to St. Mary's Hospital. 16°, cloth, 700 pp., with illustrations. Philadelphia: F. A. Davis Company, 1944. $3.50.

This is a specialized medical dictionary designed for all those interested in gynecology and obstetrics. No attempt has been made to supply a general vocabulary, but the selection of words has been limited to those dealing with the two principal subjects. Many of the definitions are encyclopedic in character, and some of the subjects are treated at considerable length. The unusual method of numbering the pages by sections, such as A-51, O-17, and so forth, is confusing and does not seem to possess any particular advantage over the usual system. The long section on the common infectious diseases of infancy and the emergencies in infancy and childhood seem to be out of place in this particular work.

Neurology of the Eye, Ear, Nose and Throat. By E. A. Spiegel, M.D., professor of experimental and applied neurology and head of the Department of Experimental Neurology, Temple University School of Medicine; and I. Sommer, M.D., lecturer in ophthalmology, Long Island College of Medicine, and consultant ophthalmologist and otolaryngologist, Chicago Eye and Ear Hospital. 8°, cloth, 667 pp., with 118 illustrations. New York: Grune and Stratton, Incorporated, 1944. $7.50.

This book is intended to cover the borderland between the field of neurology and the fields of otolaryngology and ophthalmology. The text is based on postgraduate lectures given within the last twenty-five years at the University of Vienna and at Temple University. The present publication has been based partly on the 1931 Berlin edition but has been completely rewritten. A bibliography of 1719 selected references is appended to the text. The book is well printed on good paper, with a good sight-saving type.

The Diagnosis and Treatment of Acute Medical Disorders. By Francis D. Murphy, M.D., professor of medicine and head of the department of medicine, Marquette University School of Medicine, and clinical director of the Milwaukee County General Hospital and Emergency Unit, Milwaukee. 8°, cloth, 503 pp., with illustrations. Philadelphia: F. A. Davis Company, 1944. $6.00.

This book is intended for the general practitioner and the medical student and is based on an experience of more than twenty years at the Milwaukee County General Hospital.

Operations of General Surgery. By Thomas G. Orr, M.D., professor of surgery, University of Kansas School of Medicine, Kansas City, Kansas. 4°, cloth, 723 pp., with 1396 illustrations. Philadelphia: W. B. Saunders Company, 1944. $10.00.

In this new text on general operative surgery, illustrations have been emphasized and the text has been purposely made as brief as is consistent with a step-by-step description of each operation. The chapters have been arranged, in so far as possible, according to the systems of the body or under special headings, such as plastic and neurologic surgery, urology and gynecology. Chapters on wound healing and on the treatment of fresh wounds have been included.

NOTICES

SOCIETY MEETINGS AND CONFERENCES

Calendar of Boston District for the Week Beginning Thursday, May 10

Friday, May 11

*9:00–10:00 a.m. Carcinoma of the Prostate. Dr. William C. Quimby. Joseph H. Pratt Diagnostic Hospital.

*9:00–10:00 a.m. Medical clinic. Isolation Amphitheater, Children's Hospital.

10:50 a.m. X-ray Diagnosis of Syphilis. Dr. Max Ritvo. (Postgraduate clinic in dermatology and syphilology.) Amphitheater, Mallory Building, Boston City Hospital.

12:00 m.–1:00 p.m. Clinicopathological conference (Boston Floating Hospital). Joseph H. Pratt Diagnostic Hospital.

Saturday, May 12

*10:00 a.m.–12:00 m. Medical staff rounds. Peter Bent Brigham Hospital.

Monday, May 14

*12:00 m.–1:00 p.m. Clinicopathological conference. Peter Bent Brigham Hospital.

Tuesday, May 15

*9:00–10:00 a.m. Clinicopathological conference. Drs. Chester S. Keefer and H. F. MacMahon. Joseph H. Pratt Diagnostic Hospital.

*9:00–10:00 a.m. Medical clinic. Infants' Hospital.

*12:15–1:15 p.m. Clinicoroentgenological conference. Peter Bent Brigham Hospital.

Wednesday, May 16

*9:00–10:00 a.m. Endocrine Problems in Childhood. Dr. Richard Wagner. Joseph H. Pratt Diagnostic Hospital.

*12:00 m. Clinicopathological conference. Children's Hospital.

*Open to the medical profession.

(*Notices continued on page xvii*)